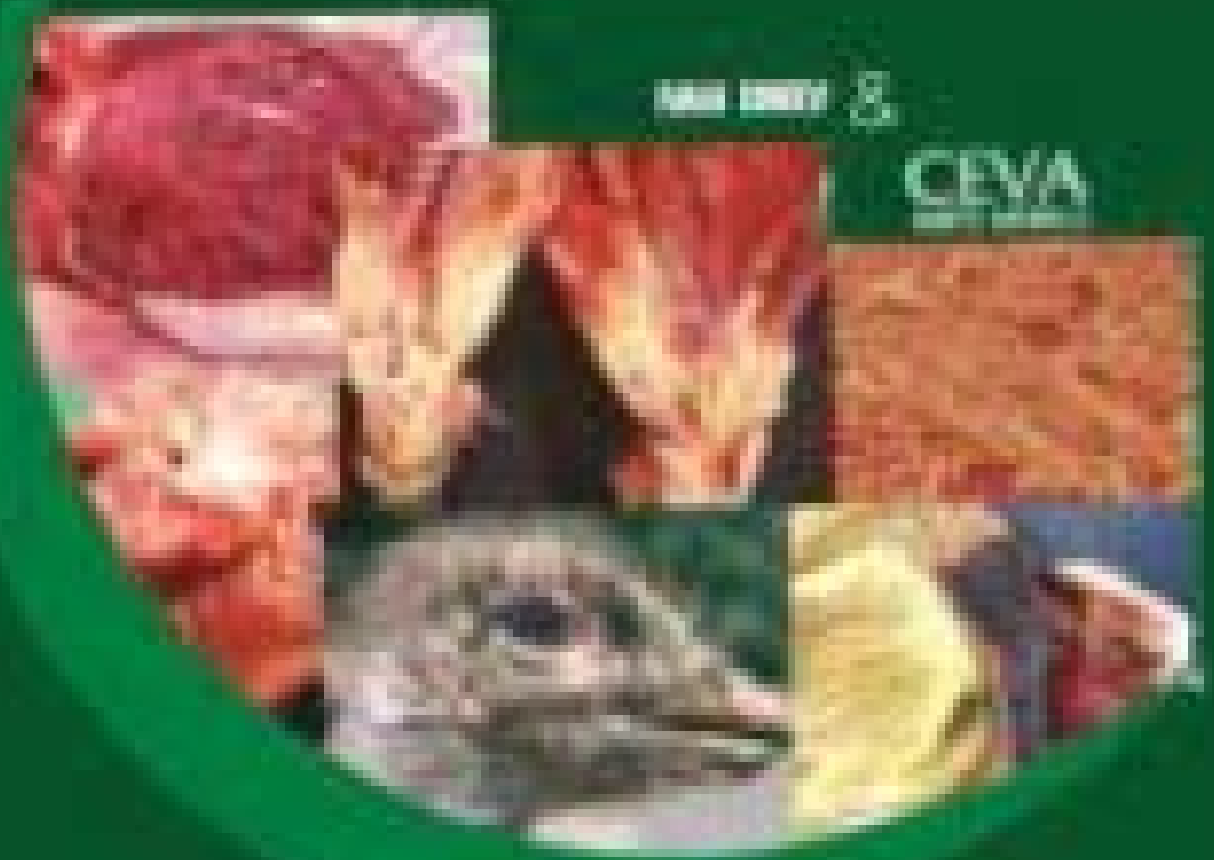
Diseases of Poultry

A COLOUR ATLAS



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CEVA

Diseases *of* Poultry

A COLOUR ATLAS

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Preface

Our primary goal in the creation of this manual was to make it helpful for the diagnostics of poultry diseases, as well as for their treatment and prevention.

Avian pathology is very rich. A new disease is discovered and identified at approximately every 4 or 5 years. The probable cause for this tendency in infectious pathology could be the high variability of aetiological agents. Although less significant, the group of non-infectious diseases is also constantly enriched. In relation to them, mycotoxicoses and some deficiency states acquired a special importance for the last few years. We can claim that the significance of neoplastic diseases is not reduced too. The usage of vaccines against Marek's disease and the creation of genetically resistant lines of birds significantly reduced losses caused by neoplastic diseases, yet the damages caused by outbreaks are still heavy. These circumstances dictate the maintenance of a high level of knowledge and experience of specialists in the field of avian health care.

The primary approach of information presentation used in this book is the photographic material, having in mind that it is the most easily accessible way to identifying lesions of the specific nosologic units. All photos in this book are original and made during our own scientific and consultation work. The atlas contains a total of 557 colour photographs, of which 503 macroscopic and 54 microscopic. The predominant share of the photographs demonstrating the gross pathology of birds is aimed at the comprehension and user-friendliness of the book. In relation to this, and with the purpose to put an emphasis on the diagnostic value of histological tests for some diseases, is the relatively low number of microscopic photographs.

The current edition includes over 50 diseases from the avian infectious pathology, and nearly the same number from the non-infectious one. Beside the classic and well-known diseases, we have included relatively new, little known or unknown until now diseases. As an example for these, we would point out cholangiohepatitis in broilers, some complications occurring after the application of vaccines contaminated with *P. aeruginosa*, haemorrhagic enteritis in turkeys, a recognized atypism for some neoplastic diseases etc. The *Aspergillus* granulomatous dermatitis, rupture of the tendon of the gastrocnemius muscle in broiler parents, rupture of the caudal renal artery in turkeys, and the influence of *Galium aparine* seeds on the health and productive indicators of broilers were included as definitely new for avian pathology. The presented case of hyperandrogenism in broilers is unique.

We hope that this edition will contribute to adding up new material in the knowledge and confidence of specialists in the field of avian diseases' diagnostics.

We expect that our book will be warmly accepted among our colleagues veterinarians, students of veterinary medicine, farmers, and all kinds of specialists in the field of poultry-breeding.

This edition also exists as an expanded electronic version, which can be used in the continuing education of specialists or beginners on the basis of wide options for discussion on the presented problems.

January, 2007
Stara Zagora

Ivan Dinev & CEVA

Acknowledgement

In the next few lines, I would like to express my gratitude to numerous friends and colleagues, whose support made the creation of this book possible.

Our idea of such an issue received the complete moral support from our colleagues as well as from farmers, specialists and poultry breeders, working in the field. It was both very pleasant and involving for us to hear the feedback from colleagues with proved experience and competence in their field.

First of all, I would like to express my personal and my family's heartfelt gratitude towards the team of CEVA ANIMAL HEALTH BULGARIA LTD, in the face of its leader, Boyan Guberkov, D.V.M. and his colleagues Bozhidar Ivanov, D.V.M., Ventsislav Mikov, D.V.M., Emil Galabov, D.V.M. and Lyubomir Malamov, D.V.M., for their cooperation and financial support in the publishing of this book. Thanks to the excellent collegial relations within the team of CEVA Bulgaria, our professional idea came to life.

I would like to specially thank Krassen Penchev, D.V.M., from „Pilko Ltd" in Razgrad, for the kindly provided photos, which are used in the atlas under numbers 175 through 185 and 496. I also thank Assoc. Prof. I. Nikiforov, PhD, for photo number 33.

Special thanks to Zheko V. Kounev, D.V.M, PhD, associate professor, Purdue University, USA, for our mutually beneficial cooperation in the professional field, and his agreement to use photographs of his under numbers 258, 261, and 362 in the book.

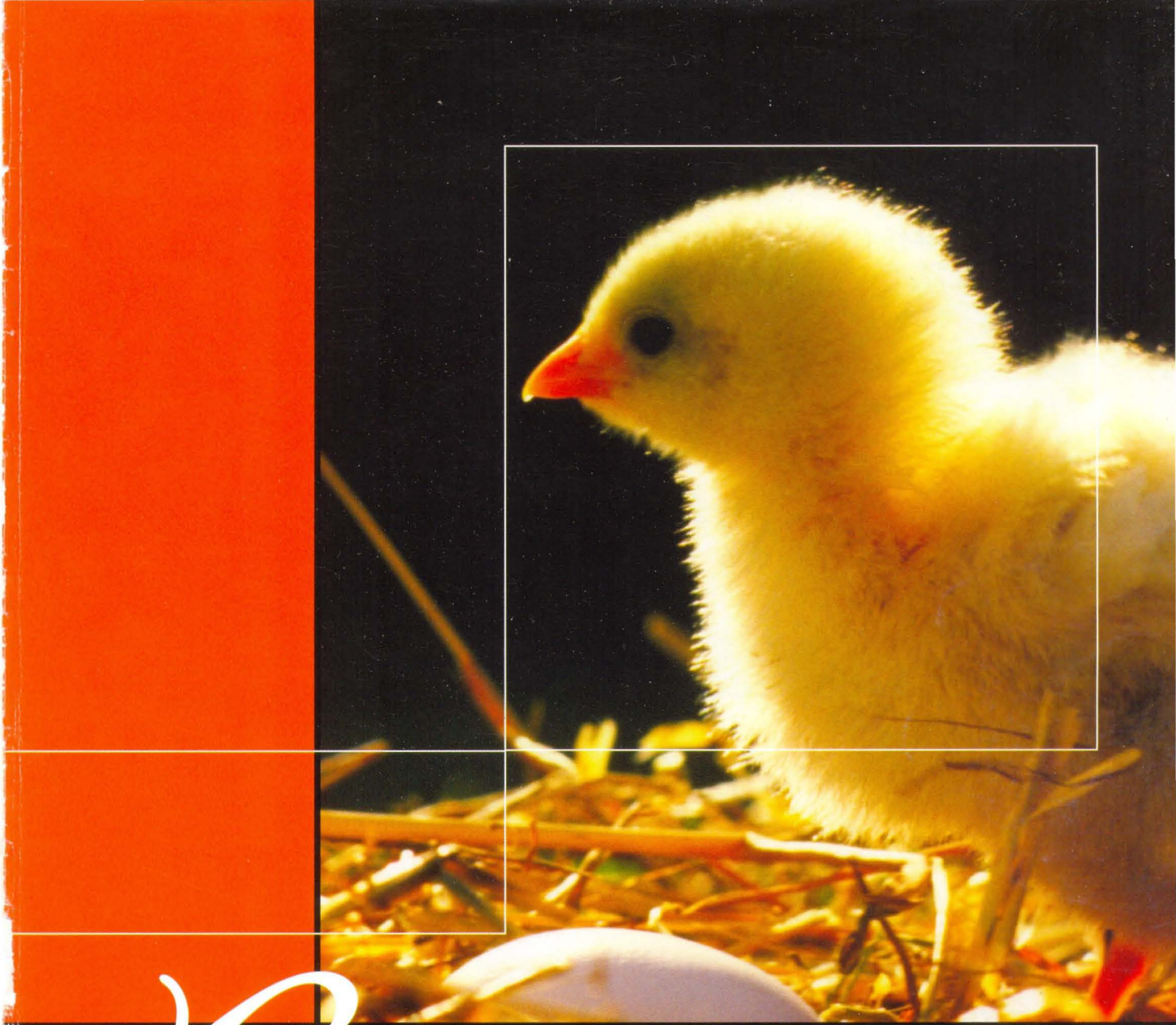
Special gratitude also to the team of the laboratory of histopathology to the Department of General and Clinical Animal Pathology at the Faculty of Veterinary Medicine, Trakia University Stara Zagora, for the perfectly prepared materials for our diagnostic and scientific work in the field of avian pathology, part of which is included as microscopic photos in this atlas.

Last, but not least, is my appreciation towards Assoc. Prof. Dr. Dimitar Stoikov, chief of Department of General and Clinical Animal Pathology at the Faculty of Veterinary Medicine, Trakia University Stara Zagora, for inspiring my interest towards avian pathology.

Thank you all who wanted this book to be accomplished.

January, 2007
Stara Zagora

Assoc. Prof. Dr. Ivan Dinev

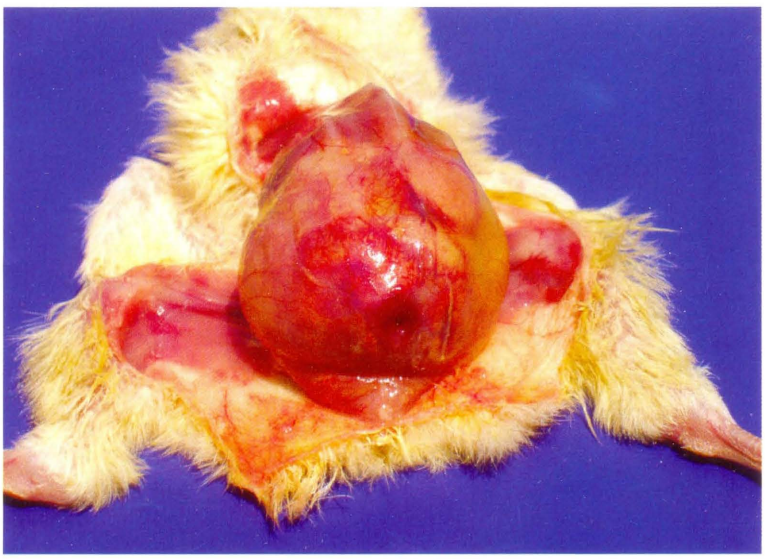


Bacterial diseases

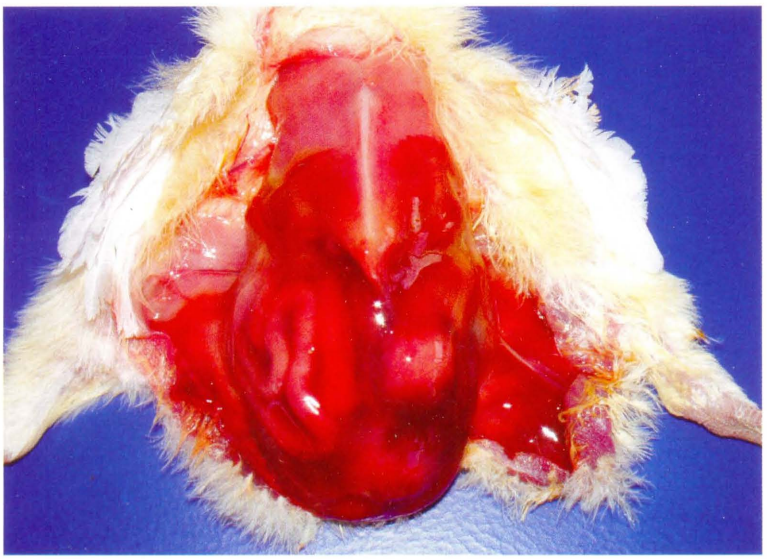
ESCHERICHIA COLI INFECTIONS

Escherichia coli infections in poultry are a group of either local or systemic diseases, where E. coli is the primary or the secondary agent.

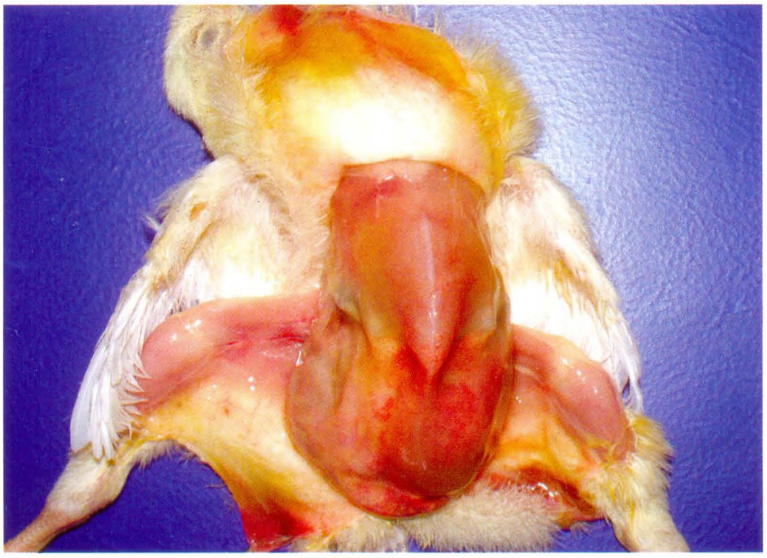
LOCAL FORMS OF E. COLI INFECTION



1. Omphalitis (navel infection). It is characterized with reddening and tissue oedema in the umbilical region.



2. *Escherichia coli* infections are widely distributed among poultry of all ages and categories. They are primarily related to poor hygienic conditions, neglected technological requirements or to respiratory and immunosuppressive diseases. A common sequel of navel infections is local or diffuse peritonitis.



3. When the amount of egg white is bigger (in larger eggs), it impedes the absorption during hatching, resulting in subcutaneous jelly-like oedemas that are an excellent media for the development of *E. coli* infections.

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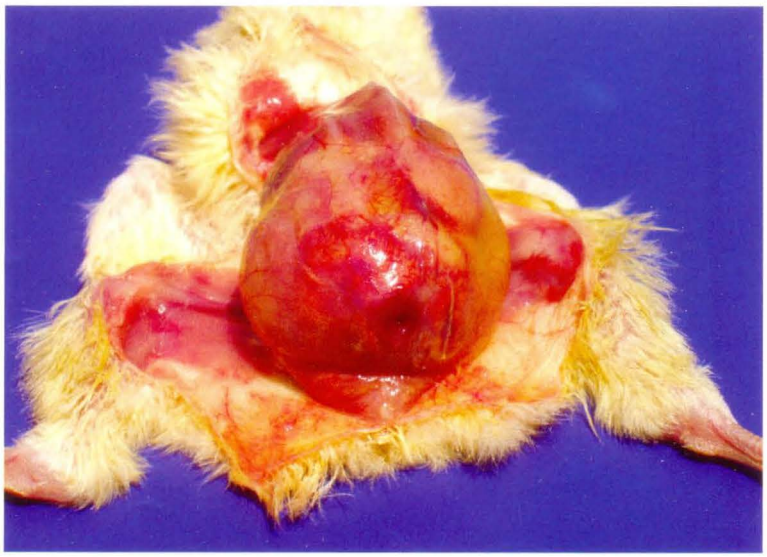

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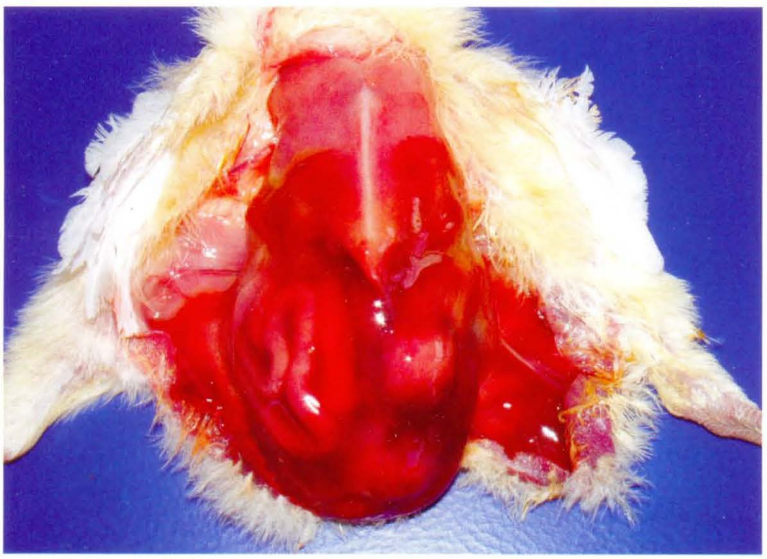

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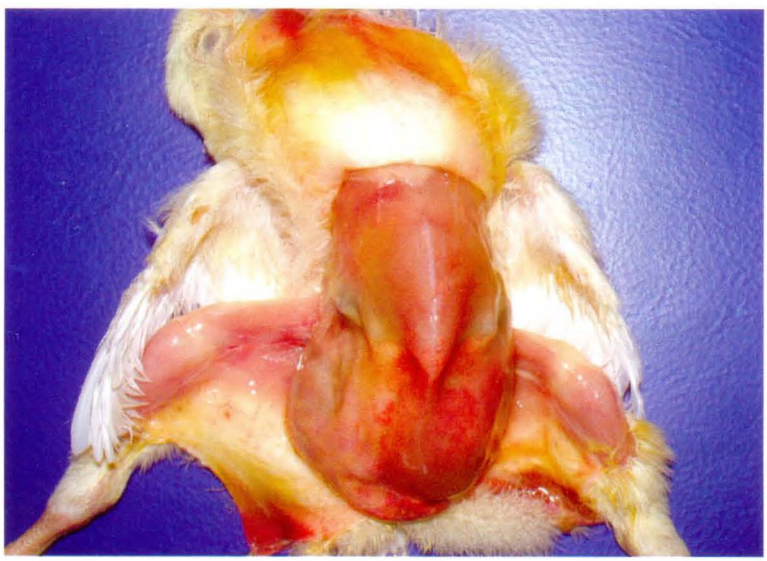

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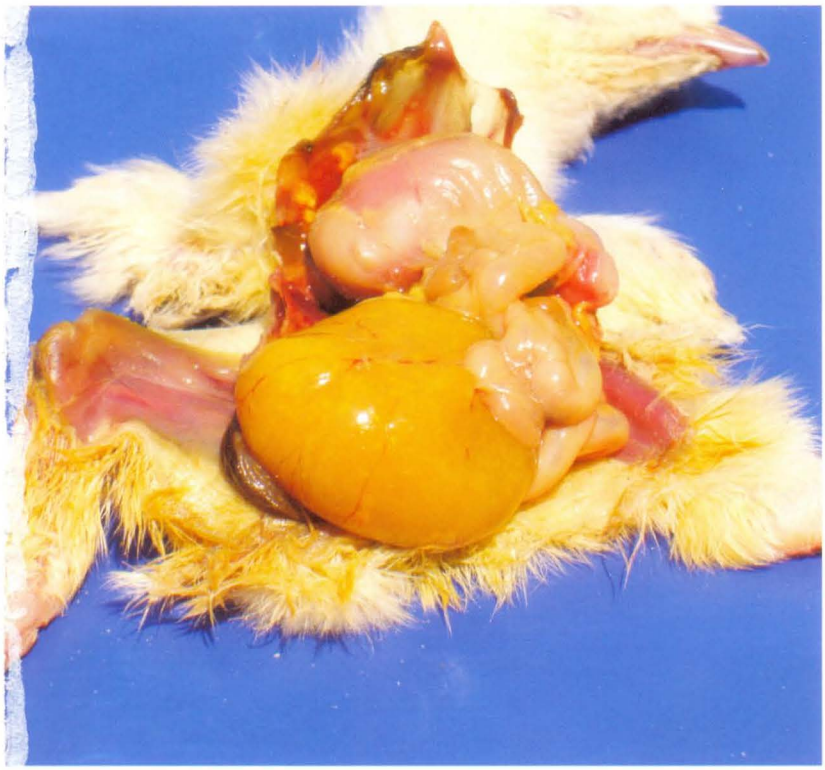
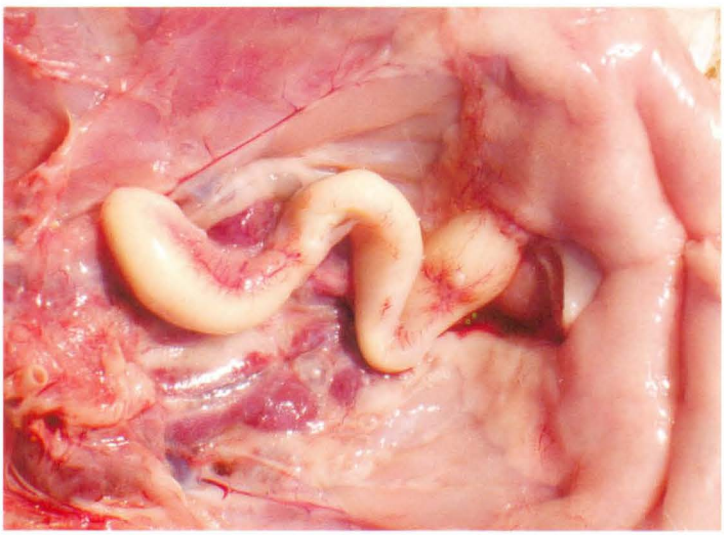


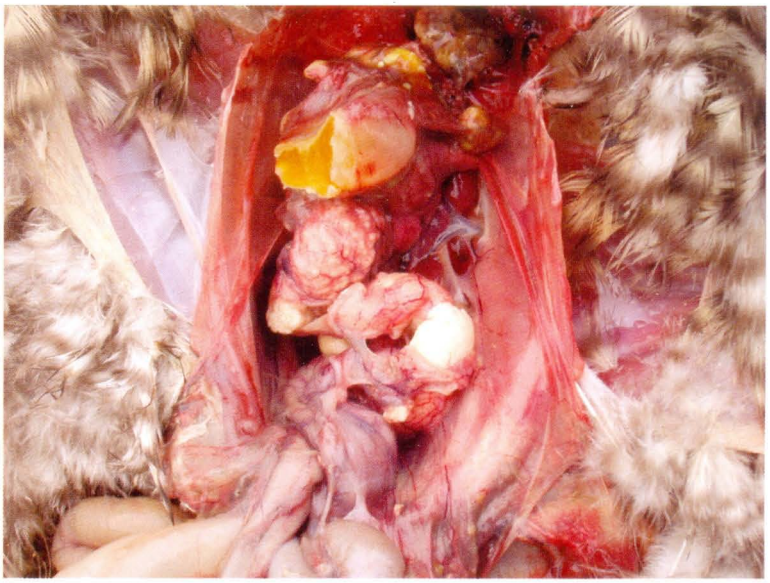
4. The delayed absorption of the yolk sac is a prerequisite for *E. coli* infections and peritonitis. The most commonly identified *E. coli* serotypes are: 01:K1 (L); 02:K1 (L) and 078:K80 (B).

5. At a later stage of the infection, the yolk content is a cause of putrefactive necrotic processes in the peritoneal cavity. The abdomen is bloated. The entire abdominal wall is affected by a moist gangrene (maceration).

6. Salpingitis (inflammation of the oviduct). Salpingites due to E. coli infections could be also observed in growing birds. The oviduct is dilated, with thinned wall and filled with caseous exudate all along its length.



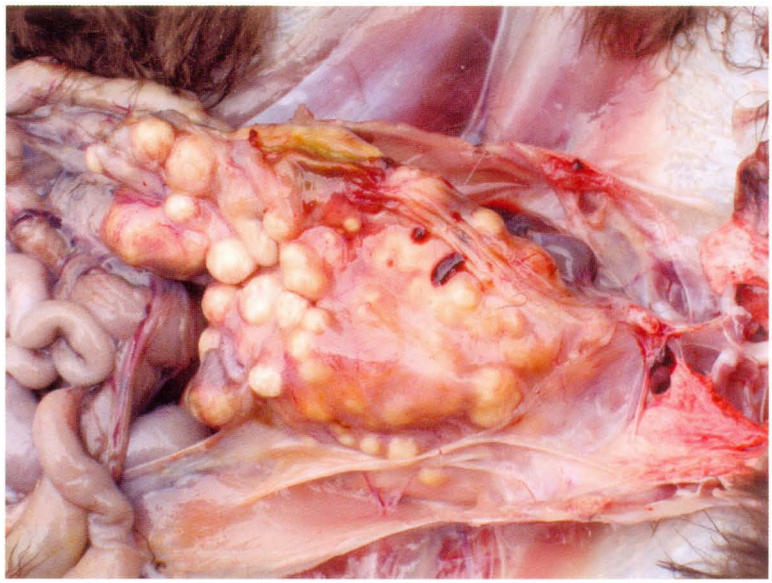
7. Salpingites are among the commonest causes for death in layer hens. *E. coli* penetrates from the cloaca via an ascendant route. Predisposing factors are the intense egg laying and the associated estrogen activity.



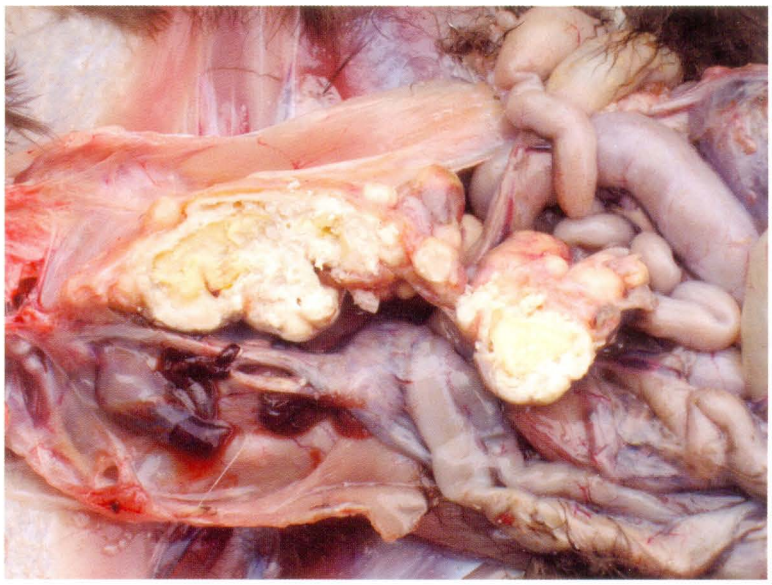
8. Salpingitis.
In older cases, the caseous masses in the oviduct have a lamellar structure. *E. coli* organisms are usually found in excreta because of their presence in avian and mammalian intestine, the birds are constantly at risk of infection through contaminated water, dust, faeces and environment.

9. Salpingitis.
Retained yolks among the caseous masses in the oviduct. In some cases, when the systemic resistance is lower, places, contaminated with *E. coli*, such as intestine, genital tract or nasal passages, could be latent sources of infection.

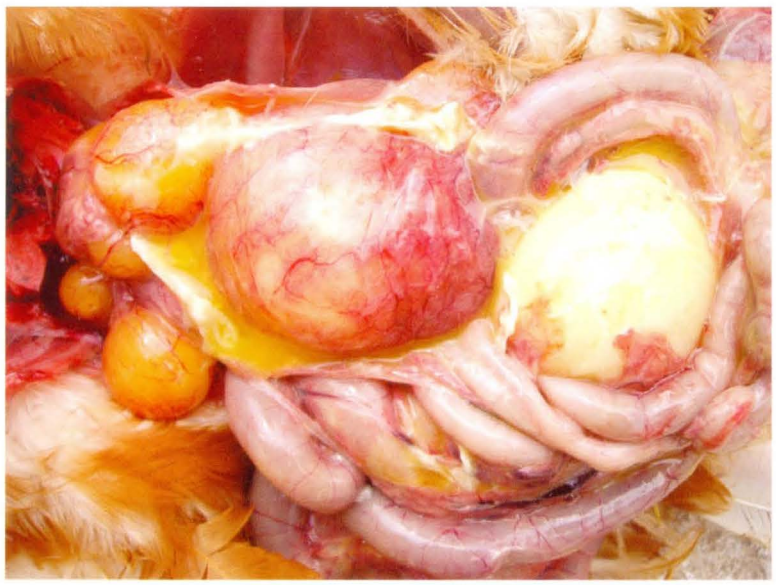
10. Salpingitis.
Compression and compactedness of caseous necrotic masses after losing a part of their water content in the oviduct of a layer.



11. Salpingitis.
An element of Fig. 10. Longitudinal cross section of the oviduct.

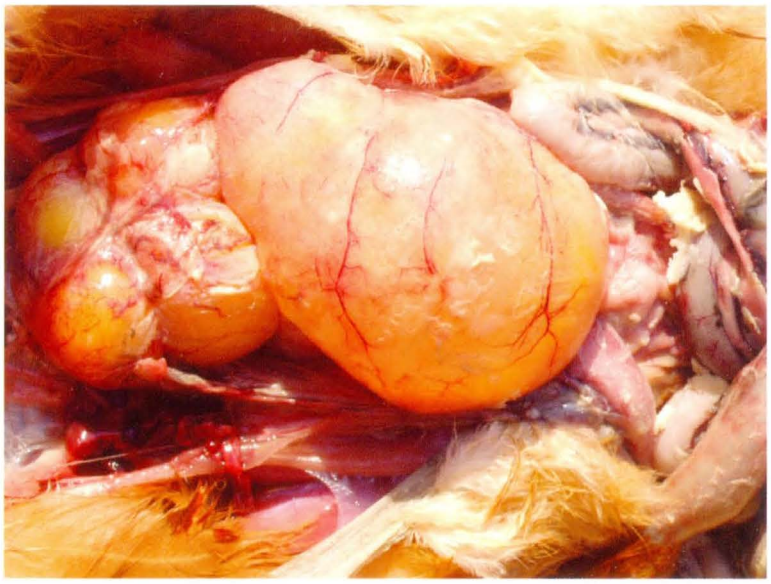


12. Egg yolk peritonitis in a layer hen consequently to *E. coli* salpingitis. The chickens could be hatched with a latent infection, when *E. coli* is present in ovaries and the oviduct. In these instances, the infection could turn into an overt infection under the influence of some stress factors or lesions.

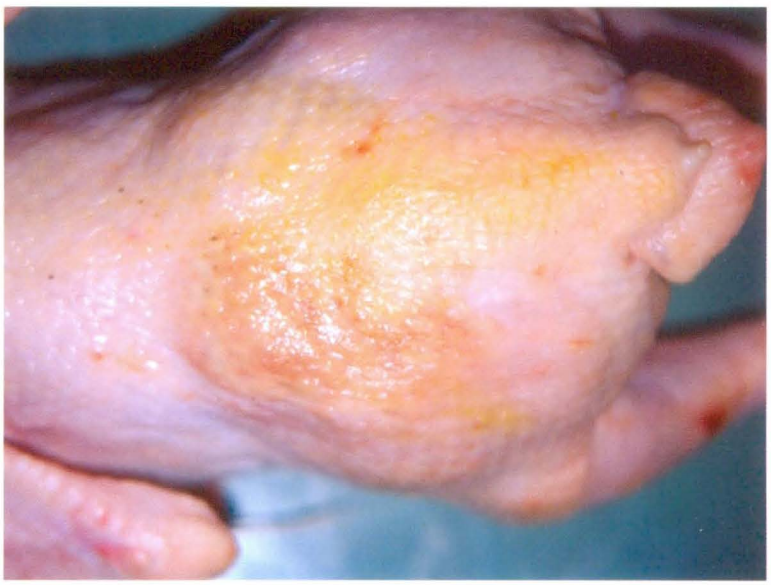


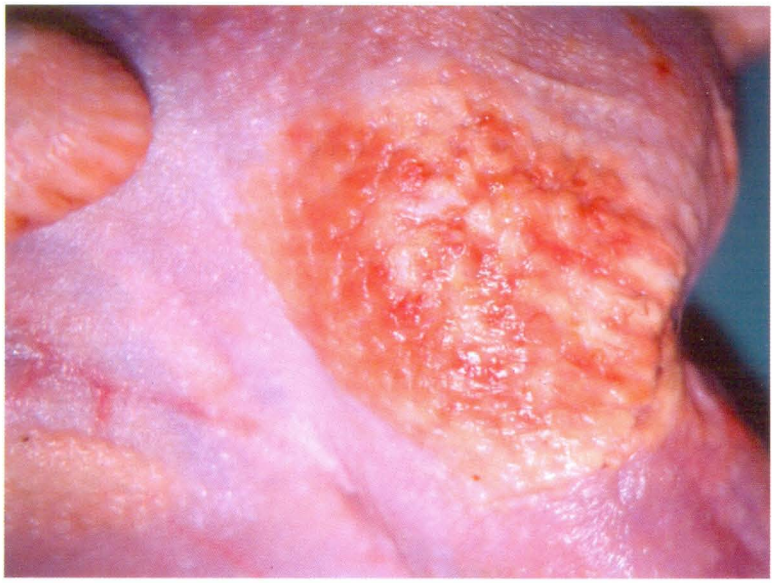
13. Oophoritis (inflammation of the ovary) consequently to a salpingitis due to ascendant *E. coli* infection

14. Cystic degeneration of ovarian follicles following an *E. coli* oophoritis.

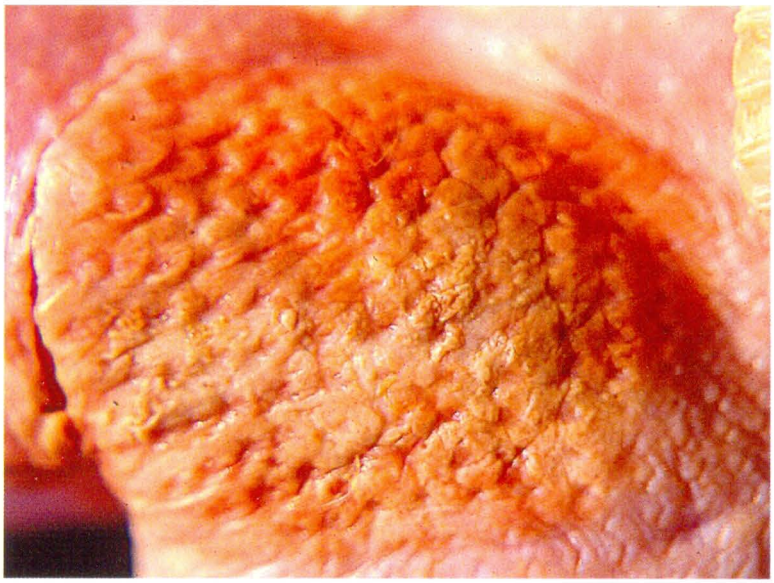


15. Cellulitis (inflammation of the subcutaneous tissue that affects also the overlying skin). It predominates in broilers and is detected mainly in slaughter-houses. Macroscopically, the lesions are with a yellowish-brown colour.



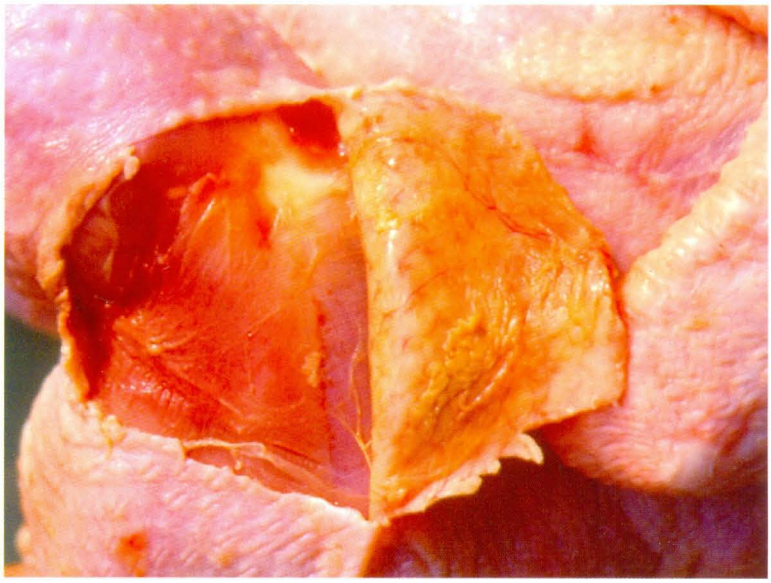


16. Cellulitis.
Affected areas are mostly in the region of the back and the thighs.



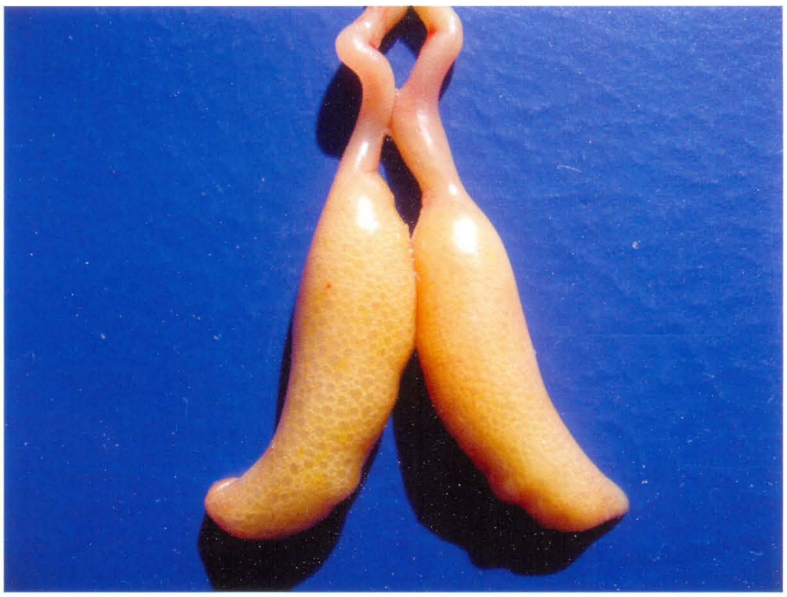
17. Cellulitis.
In some cases, the lesions are slightly prominating over the adjacent healthy skin

18. Cellulitis.
In the subcutaneous tissue, thick fibrinous plaques are often found out.

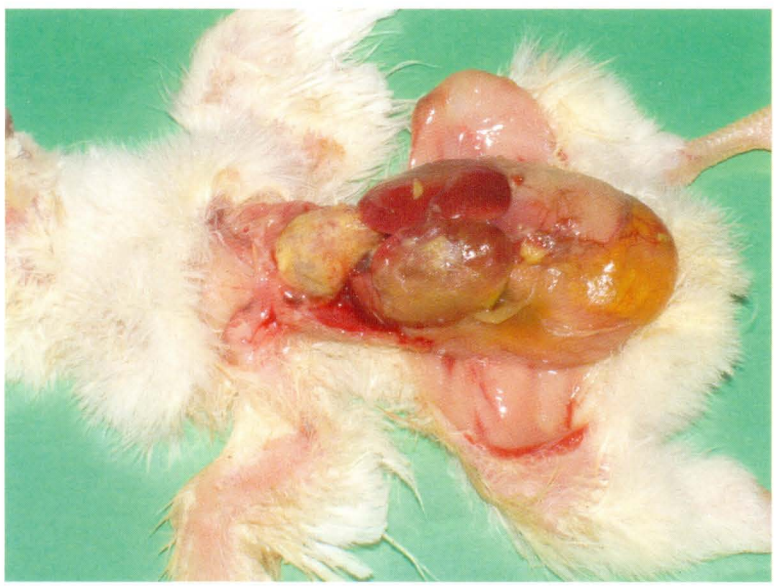


19. In some cases with adult birds, in the region of the head, subcutaneous masses of thick serofibrinous exudate resulting from a local *E. coli* infection could be detected.

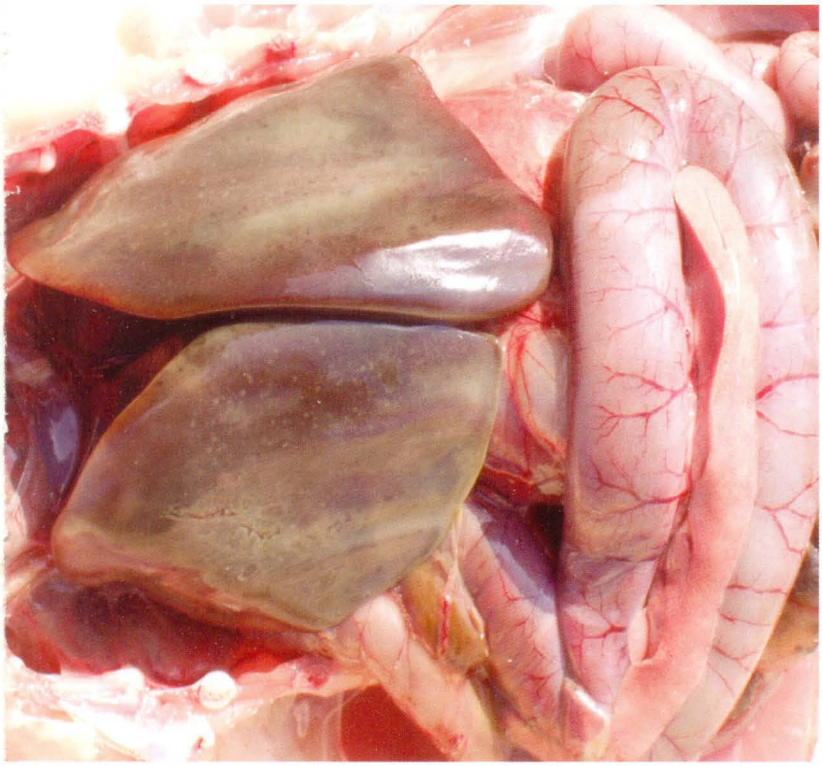
20. Enterocolitis.
Enterotoxigenic *E. coli* that produce toxins, cause the secretion and retention of fluids in some intestinal loops and especially in the caeca. Clinically, diarrhoea and dehydration are observed. The intestines are pale and distended, particularly the caeca that are overfilled with fluid containing many gas bubbles.



SYSTEMIC E. COLI INFECTIONS

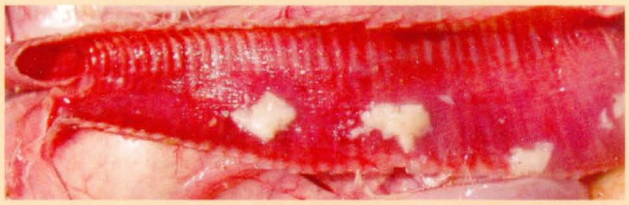


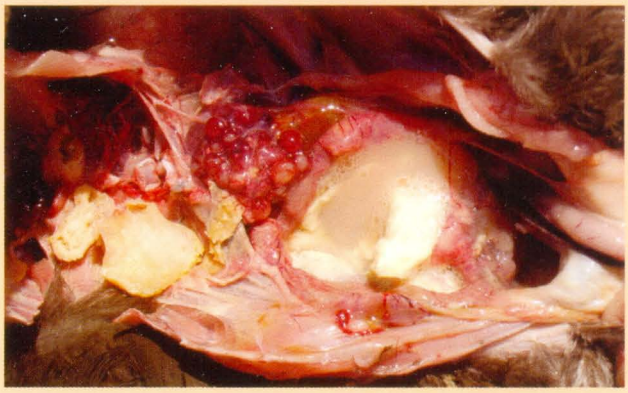
21. Neonatal *E. coli* septicaemia. Chickens in the first 24 - 48 h after hatching are affected. The death rate during the first ten days is higher and could reach 5 - 6%. The yolk sac is unabsorbed. The spleen is enlarged. Some days later, the typical serofibrinous polyserositis lesions, affecting the peritoneum, the pericardium, the air sacs and the liver capsule are manifested.

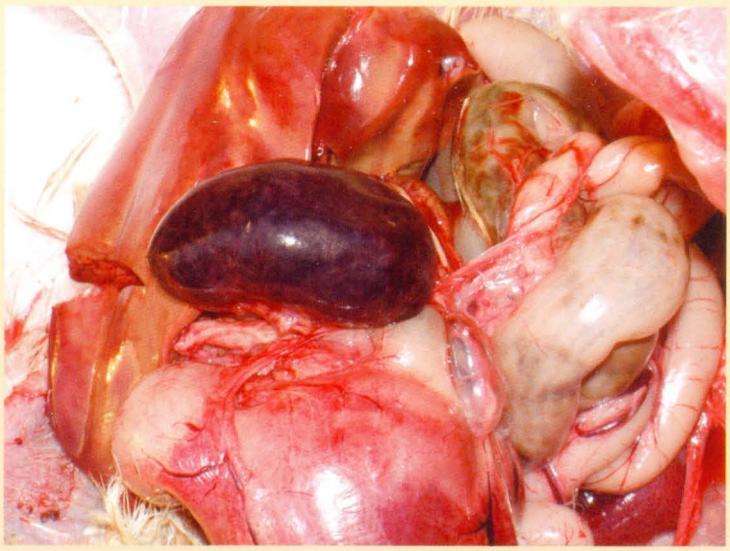


22. Acute *E. coli* septicaemia in layer hens.
Clinically and morphologically, the acute *E. coli* septicaemia could resemble fowl cholera or fowl typhoid. It is encountered in both young and sexually mature birds. The stress in the beginning of egg-laying is considered as an important predisposing factor. The parenchymal organs are enlarged and hyperaemic. Sometimes, the liver has a greenish colour and is mottled with multiple small necrotic foci. Also, pericarditis, peritonitis and petechial haemorrhages on serous coats are present.

23, 24, 25. *E. coli* septicaemia of a respiratory origin.
In such cases, the respiratory mucosa damaged by infectious and non-infections agents (ND viruses including vaccinal strains, IB, TRT, mycoplasmae, high ammonia levels) is the entrance door of the *E. coli* infection. The lesions are principally observed in the respiratory tract (trachea, lungs and air sacs), but some adjacent serous coats (pericardium, peritoneum) are also affected and thus, the picture of a typical serofibrinous polyserositis is produced.



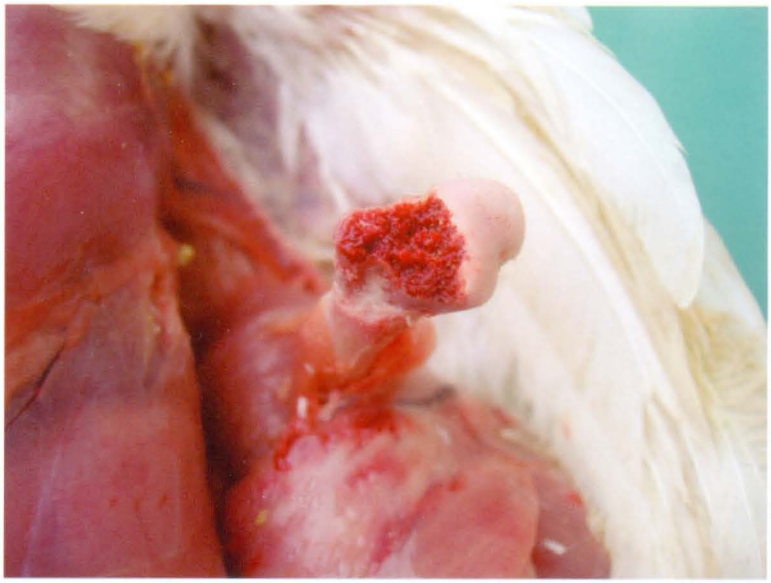


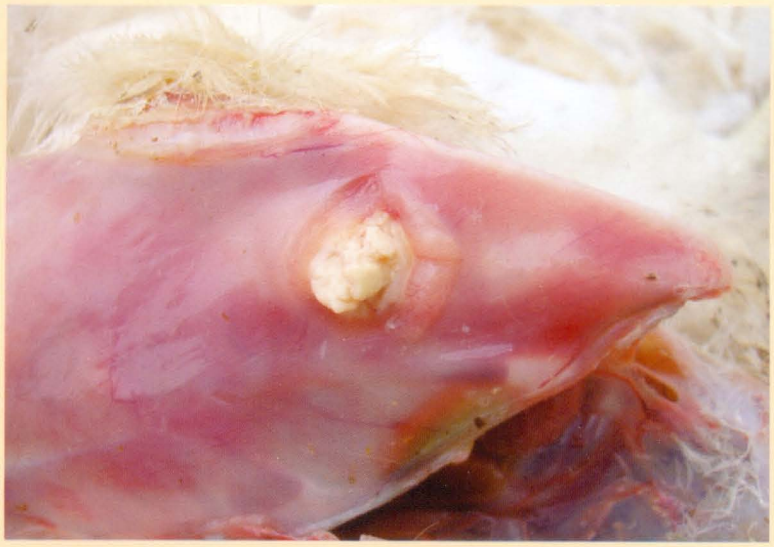


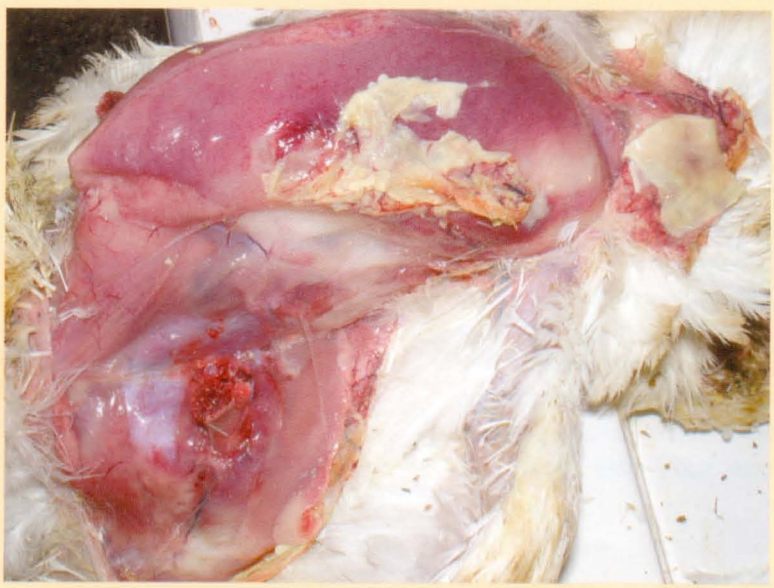
26, 27. *E. coli* septicaemia secondary to enteritis.
It is most commonly encountered in turkeys. The intestinal mucosa, damaged by the haemorrhagic enteritis virus (see Adenovirus infections), is the entrance door of *E. coli* infection. The most typical lesions are the marked enlargement, hyperaemia, haemorrhages and necrosis of the liver and the spleen.

28. Panophthalmitis (inflammation of all tissues of the eyeball). Generally, it develops secondary to *E. coli* septicaemia and is usually unilateral.

29. Arthritis, Osteomyelitis and Osteonecrosis (inflammation of joints, bone marrow and bone necrosis, respectively). The lesions are a common sequel to *E. coli* septicaemia. Clinically, lameness, prolonged lying down, dehydration and retarded growth rate are observed. The coxofemoral joints, the femur and tibiotarsal joints are most commonly affected. The bacteria colonize the physes of growing bones and provoke an inflammatory response that is further causing osteomyelitis. Pathoanatomically, fractures of the femoral head are usually discovered.

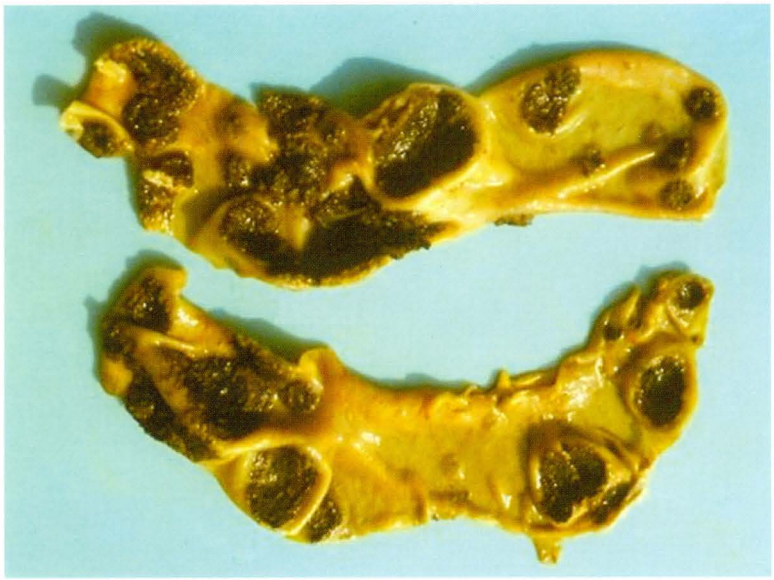






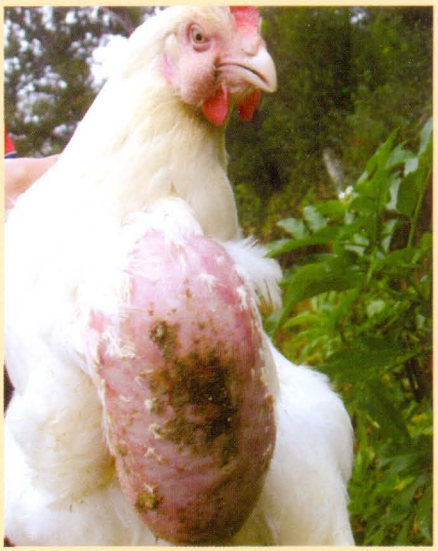
30, 31. In some cases of *E. coli* septicaemia, accumulation of exudate between the superficial and deep pectoral muscles is observed.

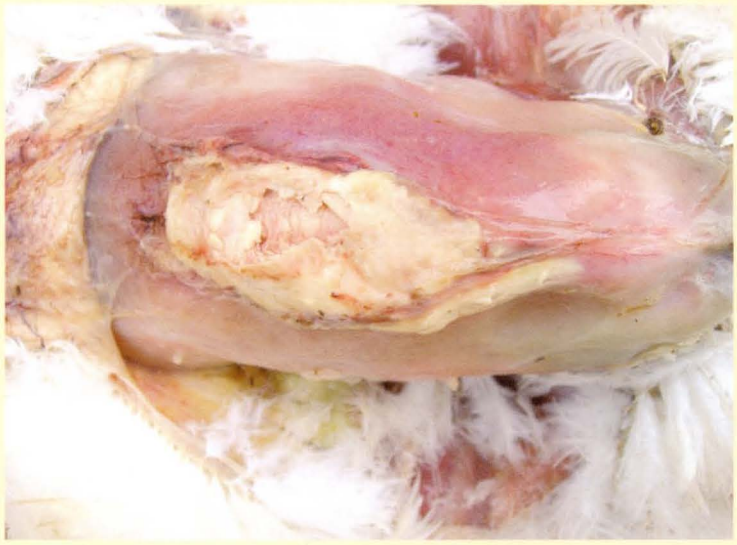
32. The lesions that develop in the articular spaces of thoracolumbar vertebrae result in spondylitis (spondylosis) and after that, in progressive paresis and paralysis.



33. Coligranuloma (Hjarre's disease). It is characterized by multiple granulomas in the intestinal tract, the mesentery and the liver, but not in spleen. The lesions are similar to these observed in tuberculosis.

34, 35. Bursitis sternalis (inflammation of the sternal bursa). The bursa is enlarged in a various extent and filled with inflammatory exudate. **The diagnosis** of coli - infections is based on isolation and typization of pathogenic *E. coli* serotypes. Many other bacteria (salmonellae, pasteurellae, staphylococci etc.), viruses, chlamydiae and mycoplasmae should be excluded as possible aetiological agents. **The prevention** should aim at minimizing the probability of faecal contamination of eggs. This implies the maintenance of clean nests, discarding floor eggs and removal of eggs that are cracked or contaminated with faeces. Breeder eggs should be fumigated or disinfected in the farm prior to their transportation in the storage premise. **The treatment** is effective if initiated soon after testing the antibacterial sensitivity of isolates.





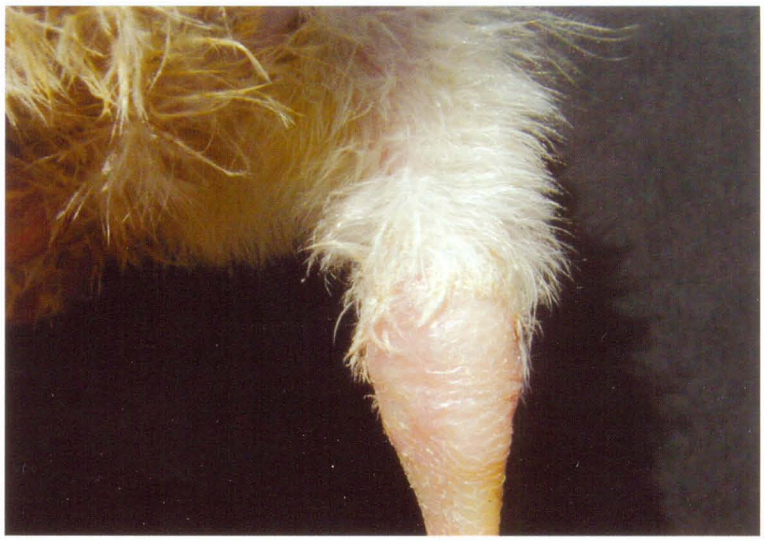
SALMONELLOSES

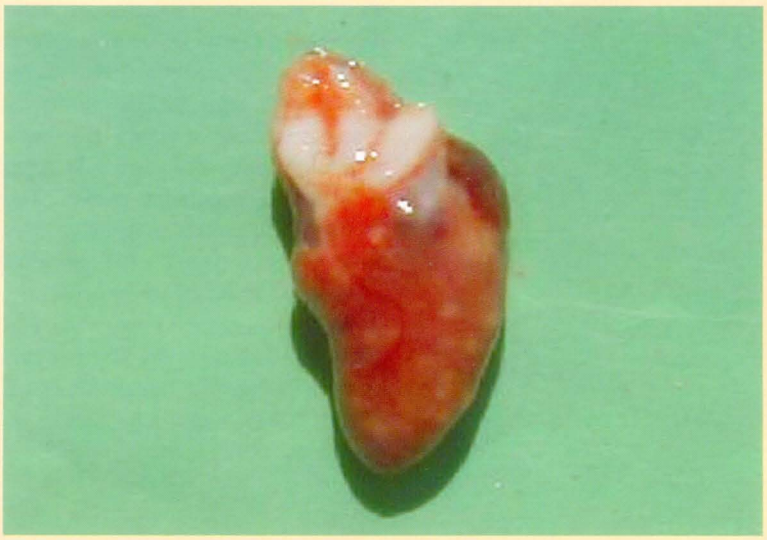
The Salmonella genus (Enterobacteriaceae family) consists of over 2400 serologically different variants (serotypes). Avian salmonelloses could be classified in two groups. The first one include infections (pullorum disease and fowl typhoid) caused by the two non-motile serotypes S. pullorum and S. gallinarum. The second group comprises infections caused by multiple motile Salmonella serotypes most frequently S. Enteritidis and S. Typhimurium isolates that are considered together as paratyphoid.

PULLORUM DISEASE

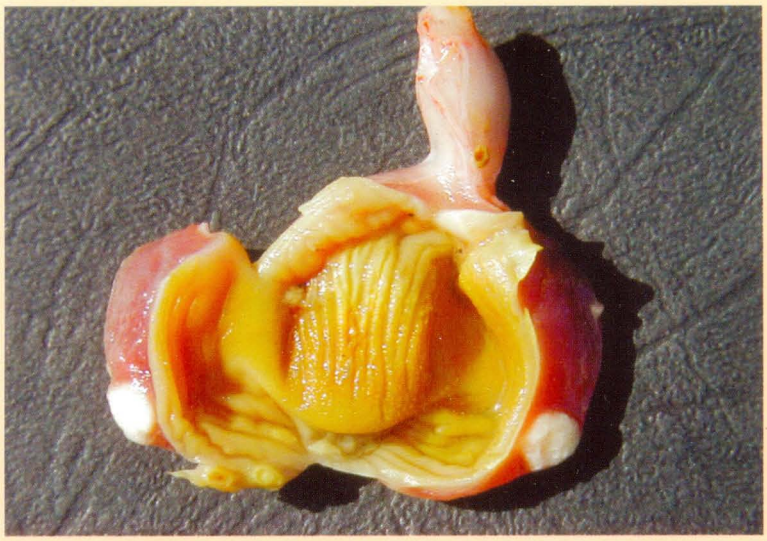
36, 37. Pullorum disease is an acute systemic disease in chickens and turkey poults. The infection is transmitted with eggs and is commonly characterized by a white diarrhoea and high death rate, whereas adult birds are asymptomatic carriers. The morbidity and the mortality rates increase about the 7^{th} - 10^{th} day after hatching. The affected chickens appear somnolent, depressed and their growth is retarded. The feathers around the vent in many chickens is stained with diarrhoeic faeces or pasted with dry faeces.

38. The oedema of tibiotarsal joints is a frequent associated sign. Pullorum disease is widely distributed among all age groups of chickens and turkeys. The highest losses are in birds under the age of 4 weeks.



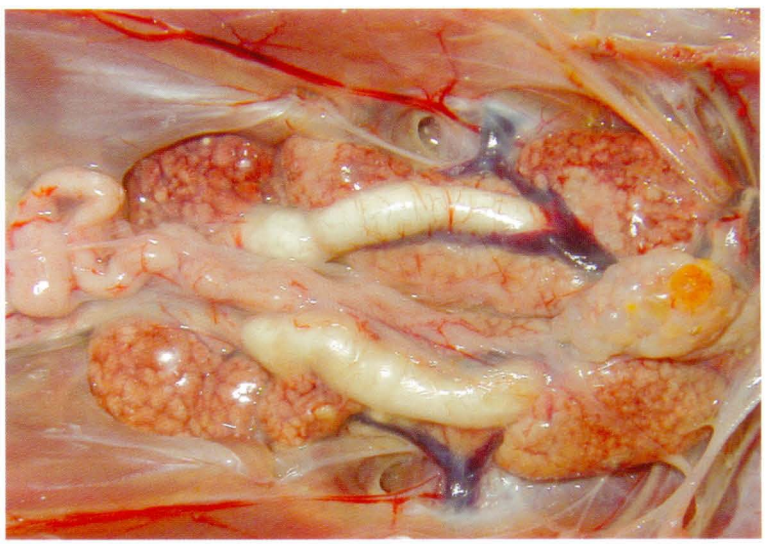


39, 40. The aetiological agent is *S. pullorum*, a non-motile Gram-negative microorganism. *S. pullorum* is very resistant under moderate climatic conditions and could survive for months. It could be killed by fumigation with formaldehyde of breeder eggs in the hatchery. Typical for this form are the greyish-whitish nodes in one or some of the following places: heart (39), lungs, liver, gizzard walls (40) and intestines, the peritoneum.



41. Sometimes, greyish-whitish milliary necroses are found out in the liver. *S. pullorum* is transmitted by infected eggs of layer hens that are carriers. Many hatched infected chickens spread the microorganism by a horizontal route to other birds via the gastrointestinal and the urinary tracts. Adult carrier birds also spread the agent through their excreta.

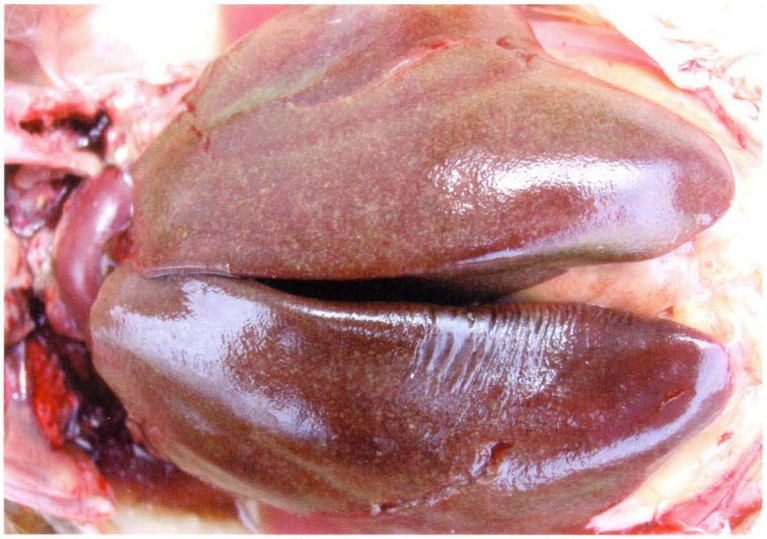
42. Ureters are often filled with urates. For confirmation of the **diagnosis**, *S. pullorum* should be isolated and typed. Pullorum disease must be differentiated from other salmonelloses, *E. coli* infections, *Aspergillus* that produces similar pulmonary lesions, *Staphylococcus aureus*, causing arthrites etc. Sometimes, the pulmonary nodes resemble the tumours in Marek's disease.



FOWL TYPHOID

Fowl typhoid is an acute or chronic septicaemic disease that affects primarily adult hens and turkeys.

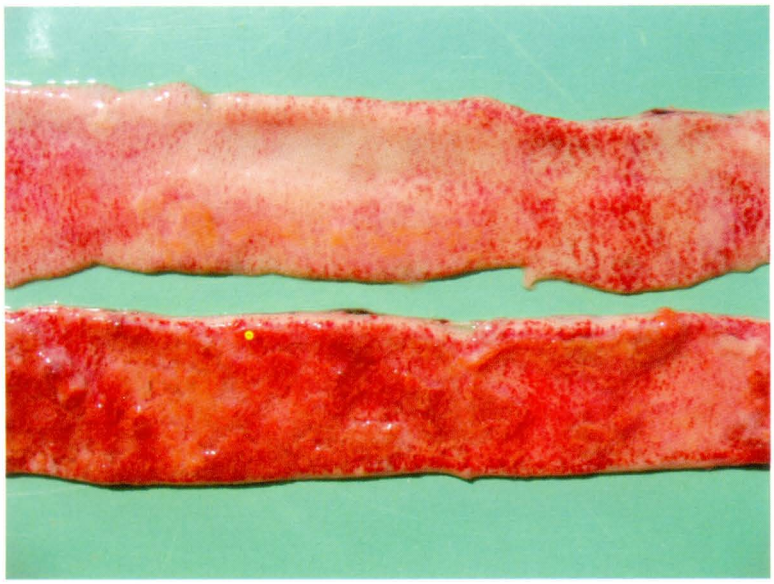
43. Acute fowl typhoid.
The outbreaks usually begin with a sharp decline in forage consumption and egg production. The fertilization and hatchability rates are considerably reduced. Diarrhoea appears. The death rate in acute fowl typhoid is high and varies between 10% and 90%. About 1/3 of chickens hatched from eggs from typhoid-infected flocks die. A characteristic lesion for acute fowl typhoid in adult birds is the enlarged and bronze greenish tint of liver.



44. Acute fowl typhoid.
In some instances, the enlarged liver is mottled with multiple milliary necroses. The outbreaks are observed primarily in hens and turkeys, but the disease is sometimes encountered in other domestic or wild fowl.

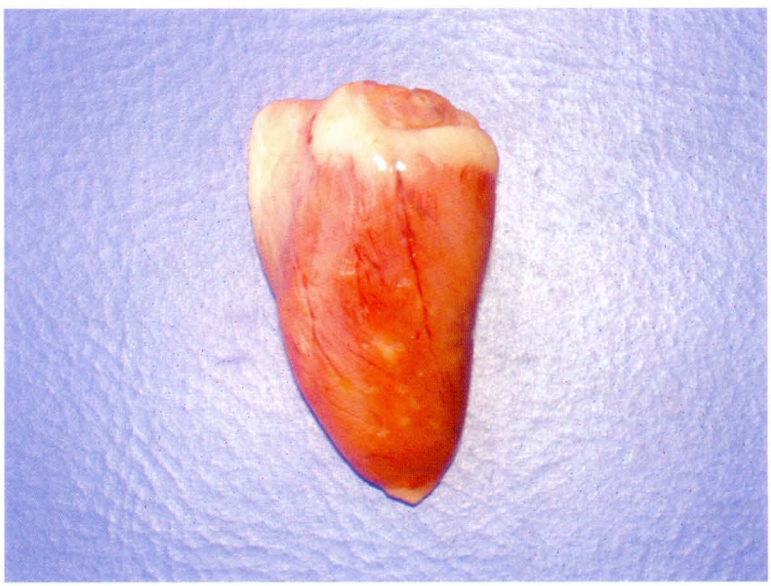
45. Acute fowl typhoid.
In other cases, the size of liver necroses varies from milliary to spots with a diameter of 1 - 2 cm. Unlike pullorum disease, fowl typhoid is lasting for months.

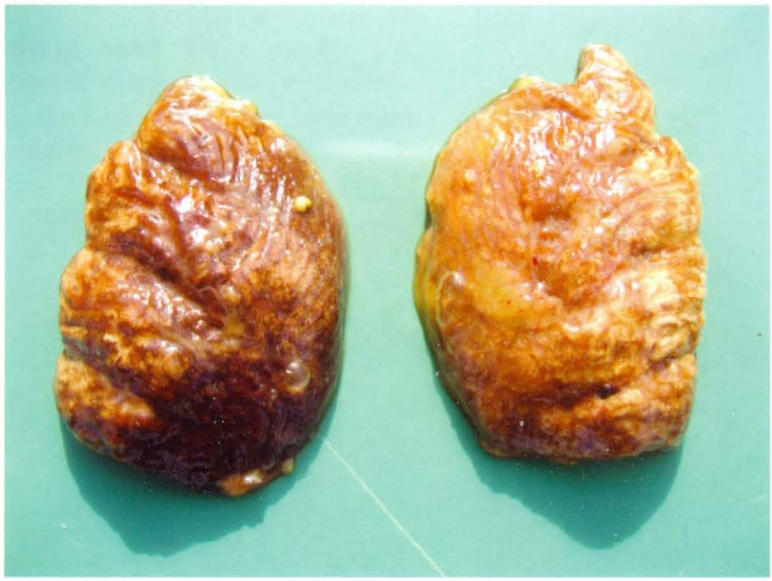
46. Acute fowl typhoid.
The spleen is 2-3 times bigger, sometimes with greyish-whitish nodules prominating on the surface, representing hyperplasic follicles.



47. Acute fowl typhoid.
Often, enteritis, especially of the anterior part of small intestine, sometimes with ulcerations, is present. The aetiological agent is *Salmonella gallinarum*. This organism usually shares common antigens with *S. pullorum* and the two microorganisms often give a cross-agglutination reaction.

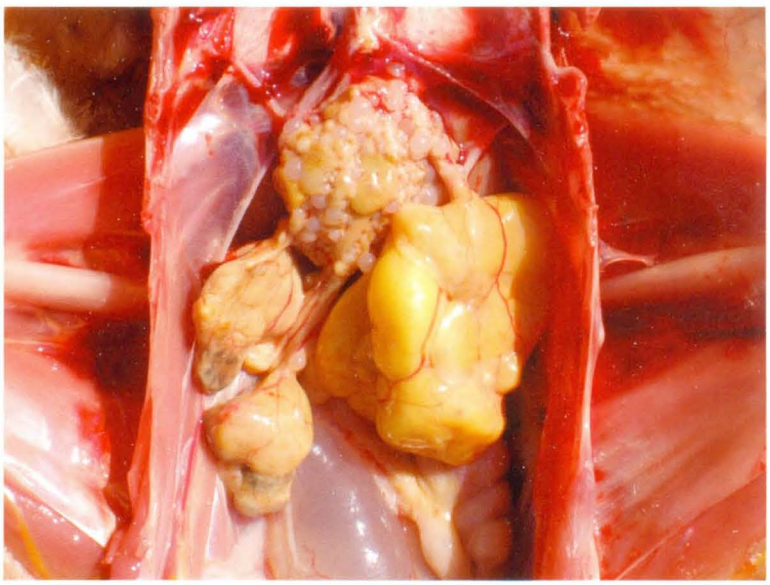
48. Acute fowl typhoid.
More rarely, myocardial necroses due to *Salmonella* toxins are detected. The transmission of the infection by contaminated eggs is especially important. Moreover, the transmission of *S. gallinarum* occurs mainly among growing or productive flocks and the death rate among adult birds is higher.

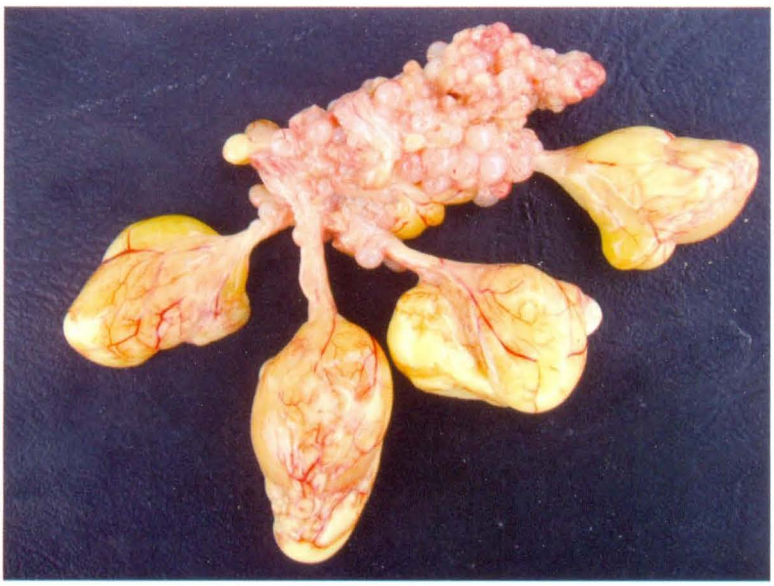




49. Acute fowl typhoid.
The lungs acquire a characteristic brown colour. Here, necroses and, following their organization, „sarcoma-like nodules" could be observed.

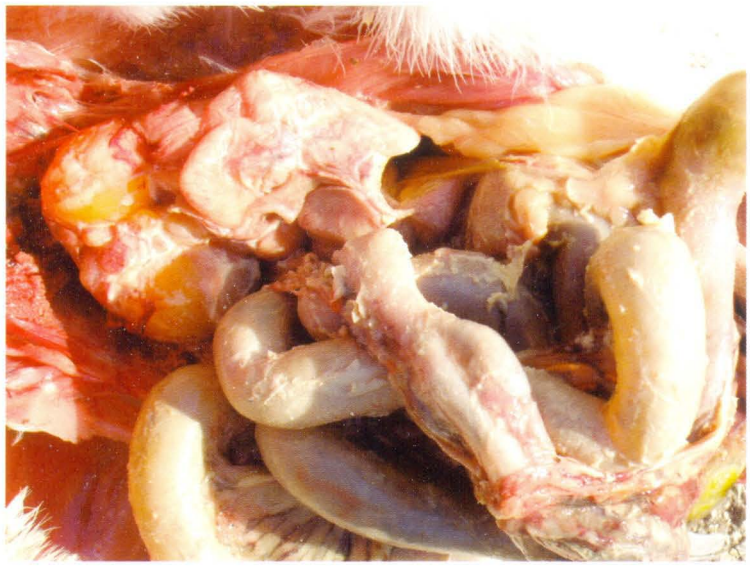
50. Chronic fowl typhoid.
The lesions are primarily in the gonads. The ovaries are affected by inflammatory and degenerative changes.





51. Chronic fowl typhoid.
Frequently, affected follicles are deformed and appear like thick pendulating masses. Fowl typhoid should be differentiated from other salmonelloses, *E. coli* infections, *Pasteurella spp.* infections etc. If breeder flocks are proved to be carriers of the infection, their eggs should not be used for breeding.

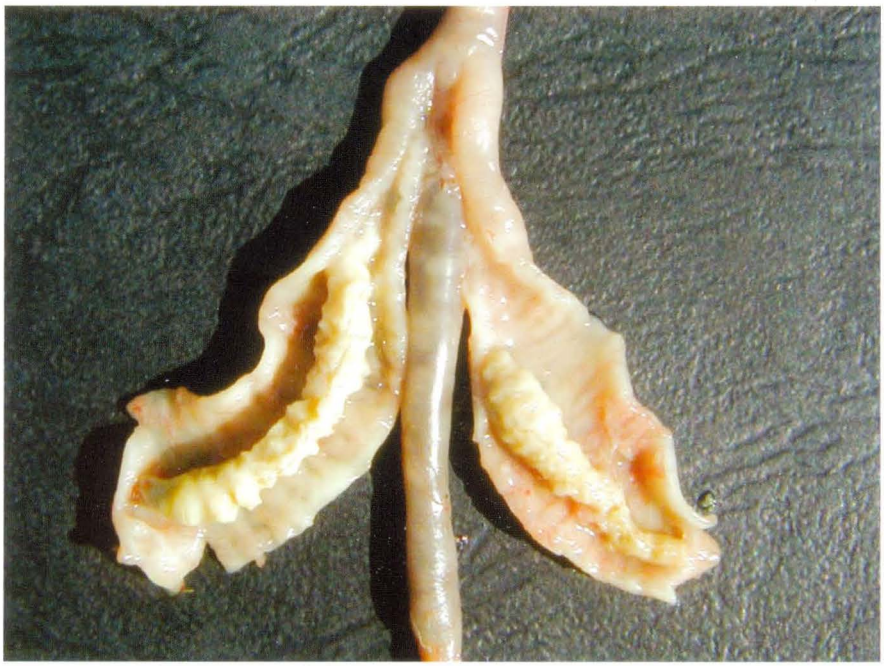
52. Chronic fowl typhoid.
Sometimes, the going out of yolk from degenerated follicles results in fibrinous adhesive peritonitis. Taking into consideration that chemotherapy does not eliminate the carriership, the treatment of poultry infected with fowl typhoid or pullorum disease is not justified and is never recommended.



PARATYPHOID INFECTIONS

53. Fowl paratyphoid is an acute or chronic disease in domestic fowl and many other avian or mammalian species, caused by some motile *Salmonella* serotypes that are not host-specific. The highest morbidity and death rates are usually observed during the first 2 weeks after hatching. The chickens are drowsy, with eyes closed, ruffled feathers and grouped near the sources of heat.

54. Diarrhoea, dehydration and pasted down appearance around the vent are observed. Pathoanatomically, marked catarrhal haemorrhagic enteritis is observed. Often the caeca are filled with gelatinous, fibrinous, cheese-like exudate. This is a finding, characteristic for salmonellosis, but it is not specific for any of serotypes.

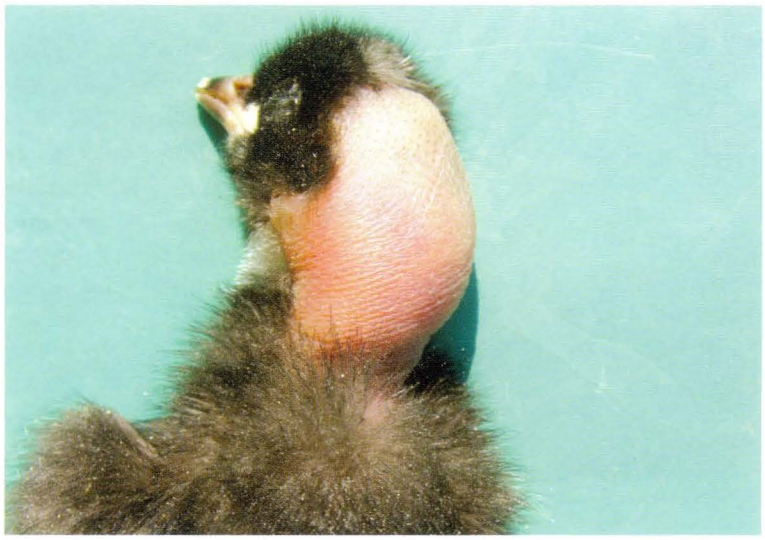


55. The inflammatory fibrinous exudate in caeca often forms casts with the shape of mucosal folds. The aetiological agents are about 10 - 15 *Salmonella* serotypes and the most common isolates are *S. Enteritidis* and *S. Typhimurium*. Most fowl paratyphoid organisms contain an endotoxin, responsible for their pathogenic effects.

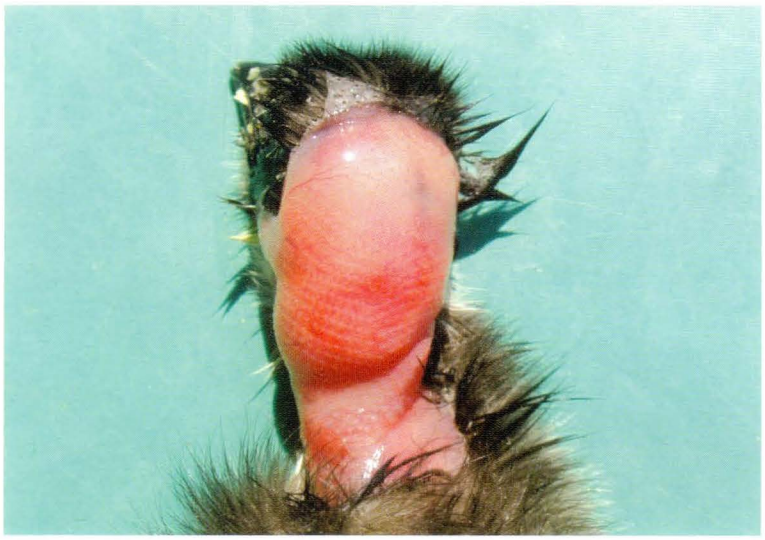
56. Sometimes, necrotic foci in the liver are discovered. The infection of small chickens occurs by penetration of microorganisms into the egg after faecal contamination. The transmission of agents could be done also by a contaminated source of animal protein (meat and bone meal etc.). The rodents are a significant reservoir of paratyphoid microorganisms. The treatment inhibits but does not eradicate the infection. The appropriate treatment minimizes the death rate until the birds develop immunity.

PSEUDOMONAS AERUGINOSA INFECTIONS

P. aeruginosa causes local or systemic infections in young or growing poultry, contaminates breeder eggs, and causes death in embryos and newly hatched chickens.

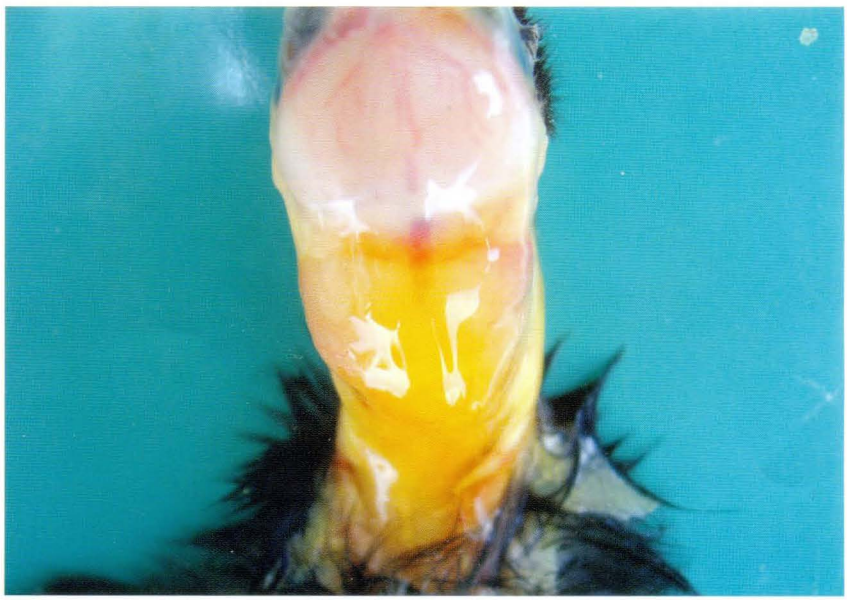


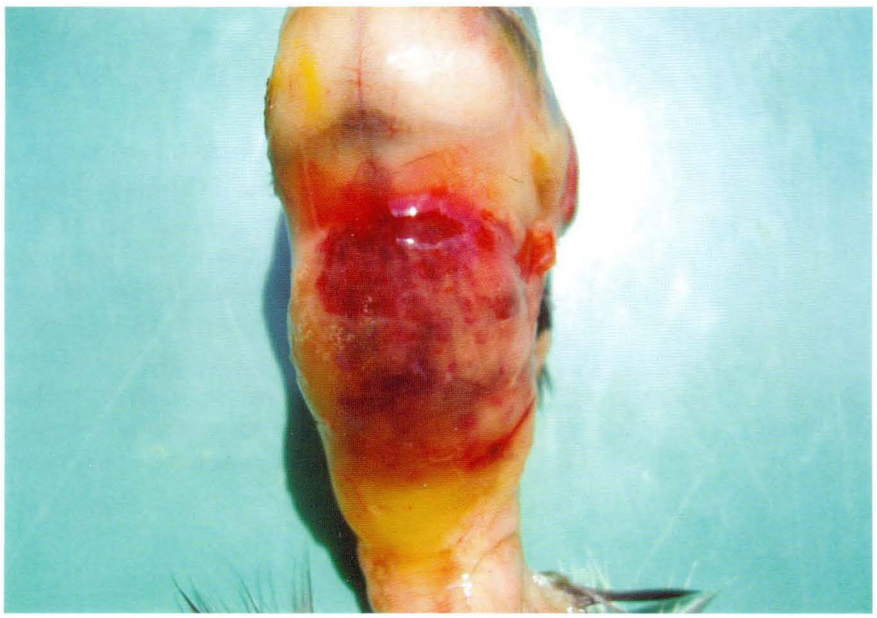
57. *P. aeruginosa* infection via a contaminated injectable Marek's vaccine. About 24h after the vaccination, nervous signs are appearing: incoordination, ataxia, unilateral lameness (if the vaccination is manual). Automatically vaccinated chickens exhibit subcutaneous oedemas in the region of the neck, sometimes involving the head.



58. The overlying skin is often macerated and the down-fallen and easily detached with the superficial epidermal layer. Most commonly, the *Pseudomonas* infection originates from hatcheries.

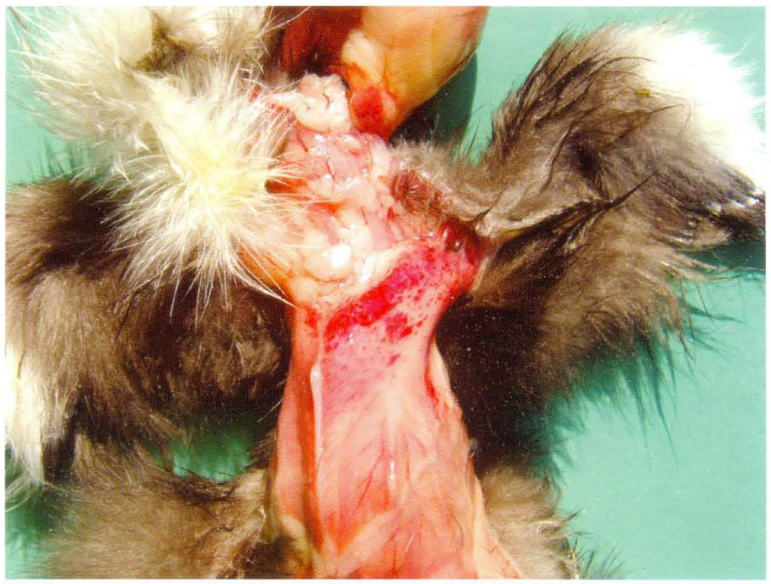
59. Subcutaneously, serous or haemorrhagic oedemas are detected. *P. aeruginosa* is a motile, Gram-negative, non-sporing rod. The microorganism is an aerobe, grows on ordinary bacteriological media and produces a green pigment composed by fluorescein & pyocyanin that has a characteristic fruity odour.



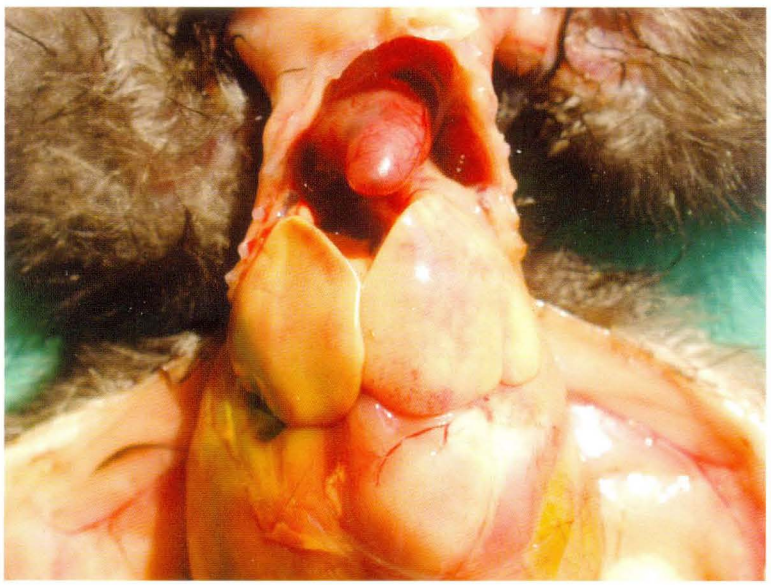


60. A subcutaneous haemorrhagic oedema in the region of the neck about the site of MD vaccine application.
P. aeruginosa is widely distributed in the soil, water and the environment. The high humidity favours its development. Susceptible avian species are chickens, turkeys, pheasants, ducks, goose, ostriches and exotic birds.

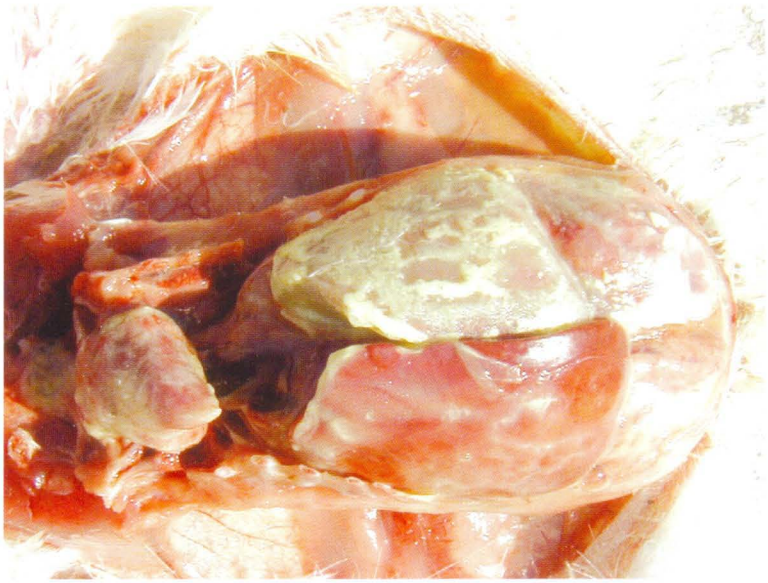
61. Sometimes, subcutaneous haemorrhages in the muscles are present.

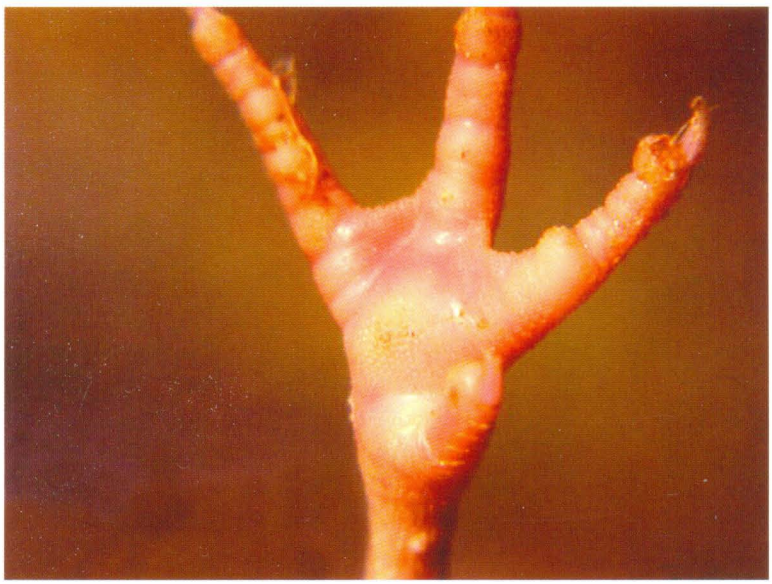


62. In the liver, hyperaemia, subcapsular haemorrhages and dystrophy are detected.

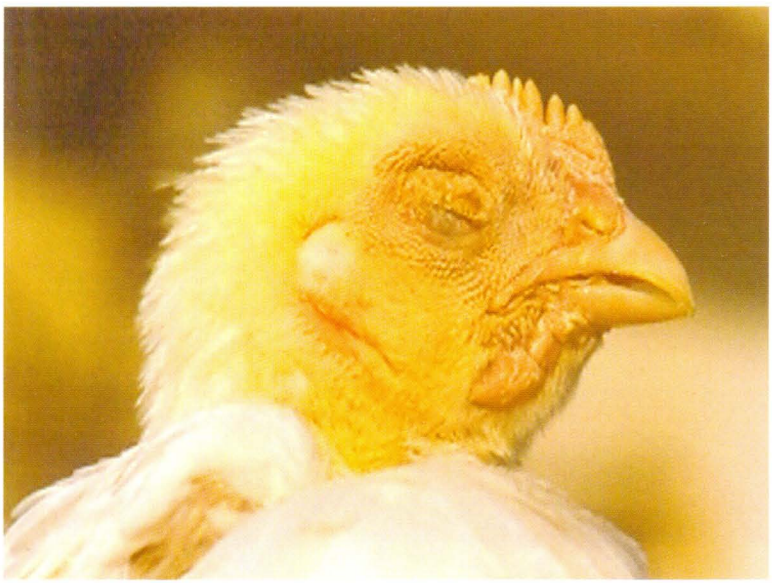


63. Acute septicaemic *P. aeruginosa* infection.
Serofibrinous inflammation of serous membranes in the pleuroperitoneal cavity (aerosacculitis, pericarditis, and perihepatitis) is detected. The lesions strongly resemble (imitate) these observed in *E. coli* septicaemia.



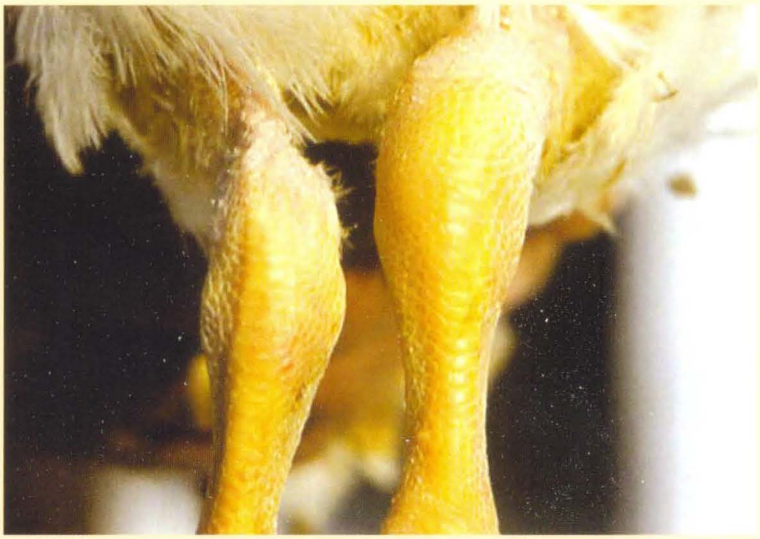


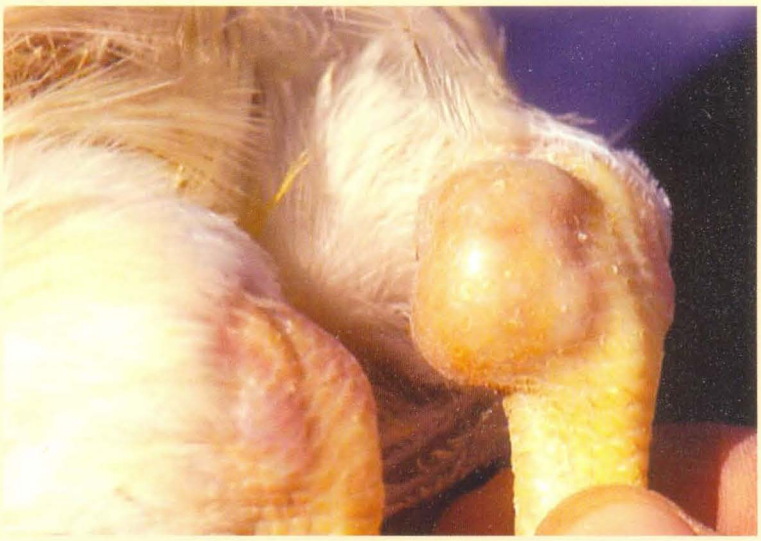
64. Local *P. aeruginosa* infections. They are encountered secondary to septicaemia or independently. Pododermatitis and inflamed footpad are usually observed in broilers at the age of 7 - 14 days.



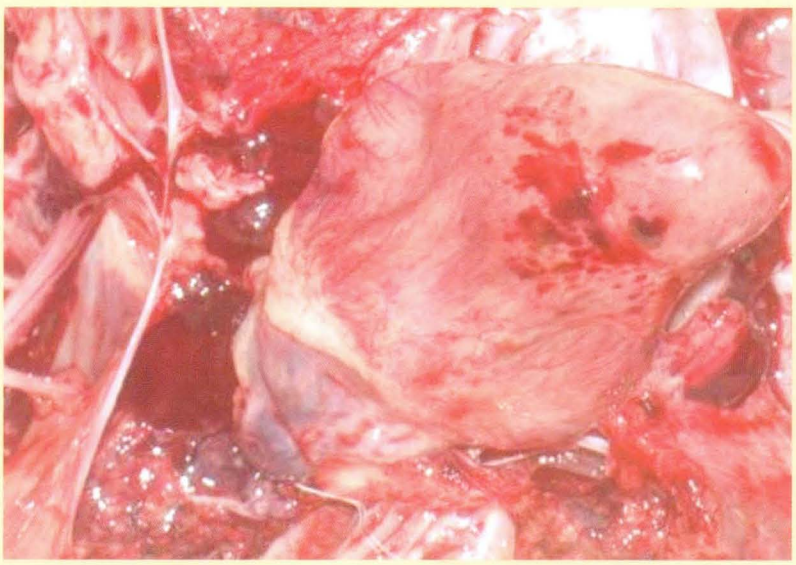
65. Other local forms of *P. aeruginosa* infection are conjunctivites and consequent keratites and panophthalmites.

66, 67. Arthrites and periarthrites are encountered in broilers as well as in growing birds. Tibiotarsal joints are most commonly affected. The skin and the mucous coats are the entrance door of the infection. The prevention is based upon detection and elimination of the causative agent. The strict hygiene in hatcheries and throughout the injection of birds is essential for the prevention of Pseudomonas infection. The test of isolates' sensitivity is of most importance for the treatment, because the microorganism is resistant to a high number of antimicrobial drugs.

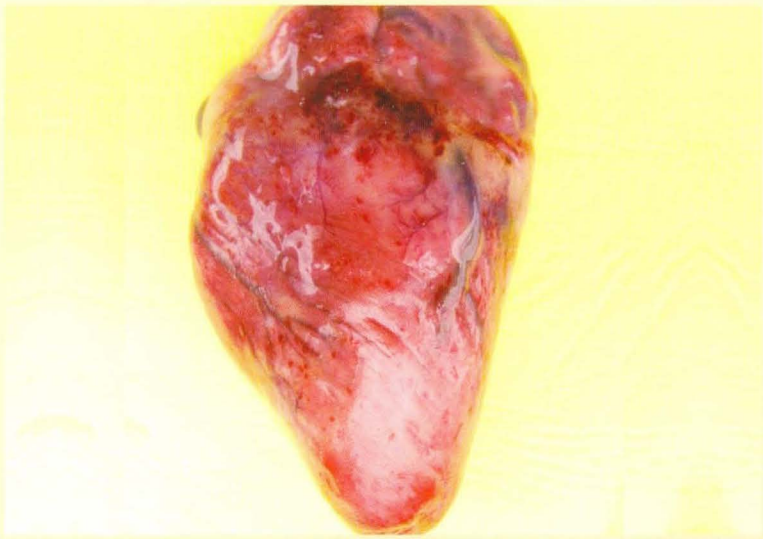




FOWL CHOLERA

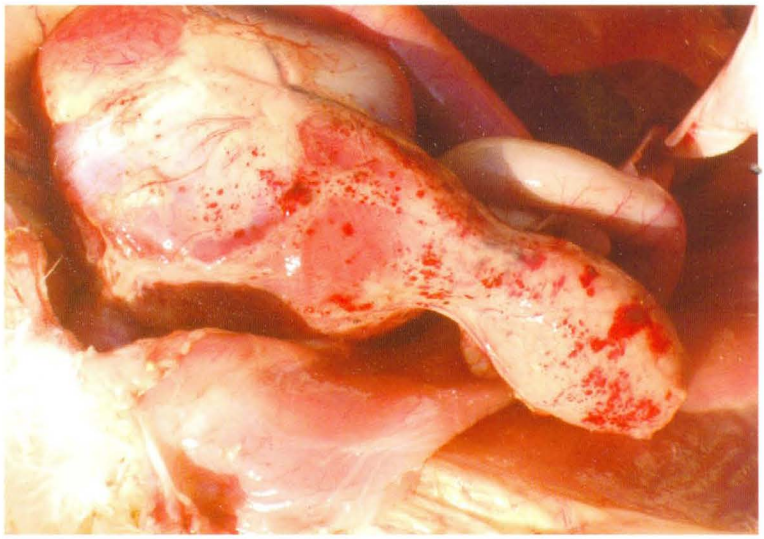


68, 69. Fowl cholera is an infectious disease in domestic fowl, waterfowl and other avian species. It is manifested either in acute septicaemic form with a high morbidity and death rates or as chronic local forms (independently or secondary to acute ones). **Acute fowl cholera.** The sudden and unexpected death could be the first sign of the disease. In this form, the lesions are predominantly related to vascular injuries.



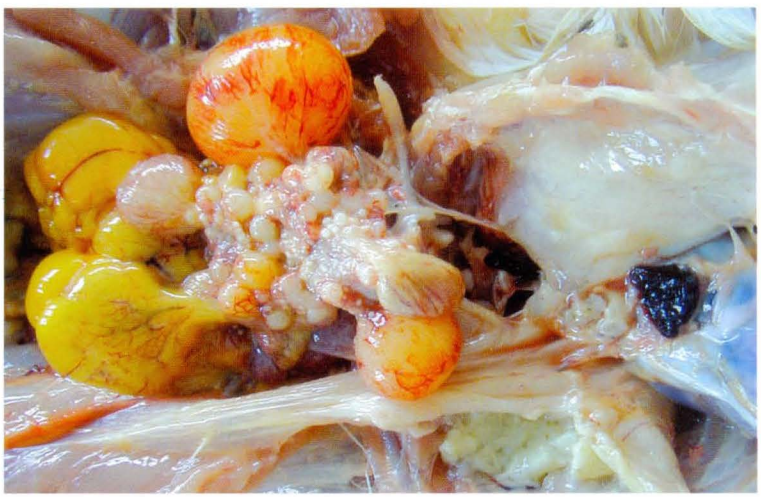
The multiple subepicardial petechial haemorrhages affecting the heart are a characteristic finding.

70. Commonly observed signs are anorexia, ruffled feathers, oral and nasal mucus discharge, cyanosis and white or greenish watery mucoid diarrhoea. Frequently, subserous petechial or ecchymosed haemorrhages in the anterior part of the small intestine, the gizzard or the abdominal fat are discovered.



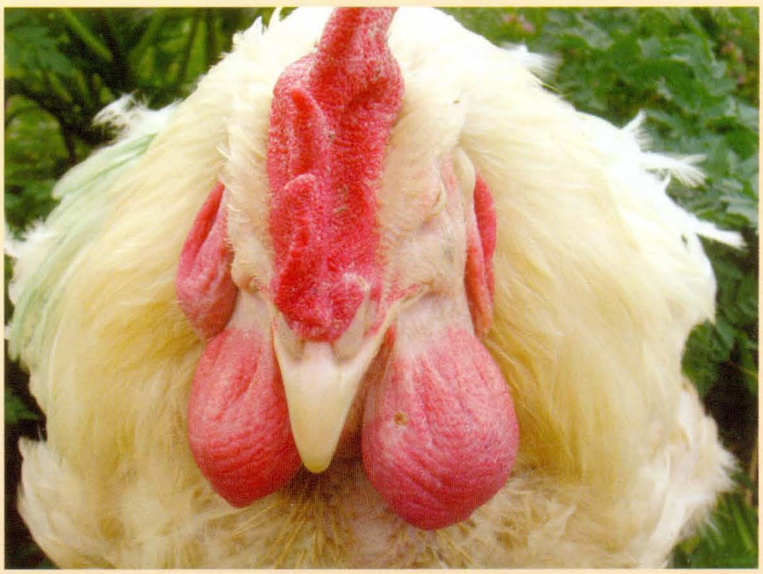
71, 72. In the liver, multiple milliary or submilliary necroses are present. Fowl cholera is encountered sporadically or enzootically, sometimes with a high mortality, sometimes yet the losses are insignificant. The disease prevails during the late summer, the autumn and the winter. The affected birds are in the period of sexual maturation or older. The aetiological agent is *Pasteurella multocida*, a Gram-negative micro-organism with bipolar staining, growing easily on blood agar, but not on McConkey's agar. The virulence of isolates varies considerably. *P. multocida* is easily killed by routine disinfectants.

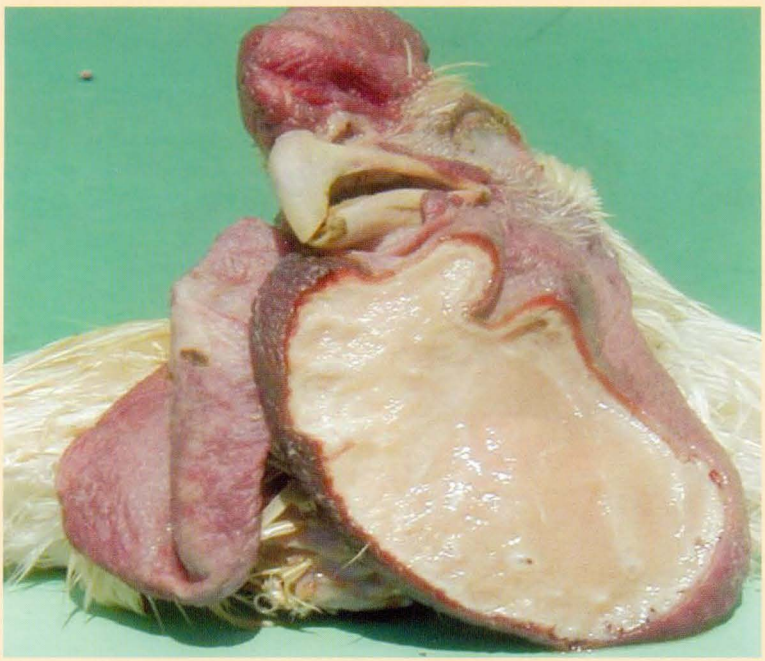
73. In layers (commercial or breeders), acute oophorites with regressing follicles and consequently, diffuse peritonites are commonly observed.

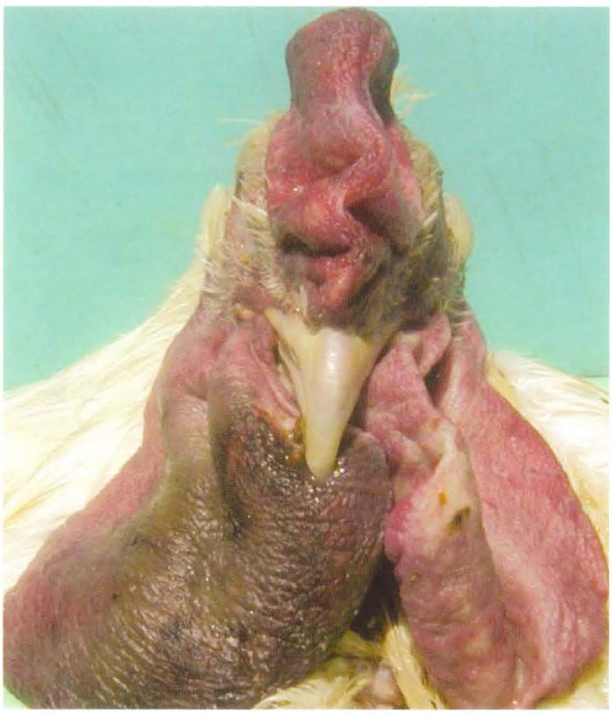


74. Chronic fowl cholera.
It is characterized by local inflammations. The periorbital sinuses are frequently affected by a serofibrinous inflammation.

75, 76, 77. Another local form is the injury of wattles that are strongly distended because of their filling with fibrinous caseous content. The flocks that recuperated from fowl cholera continue to carry and shed *Pasteurella multocida*. The carriers store the organism in nasal choanas and contaminate the forage, water and the environment with oral discharges. Wild birds and some mammals (swine) could also carry the agent and introduce it into poultry flocks. Cannibalism is an essential route of spreading the infection.

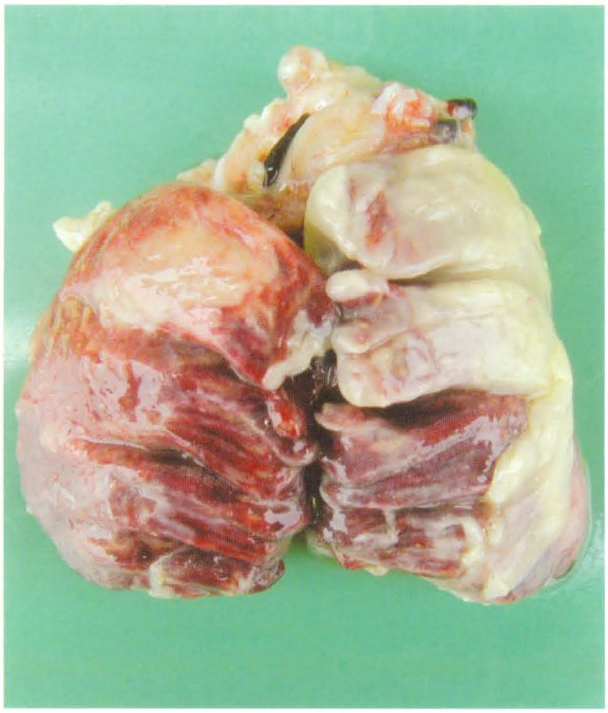


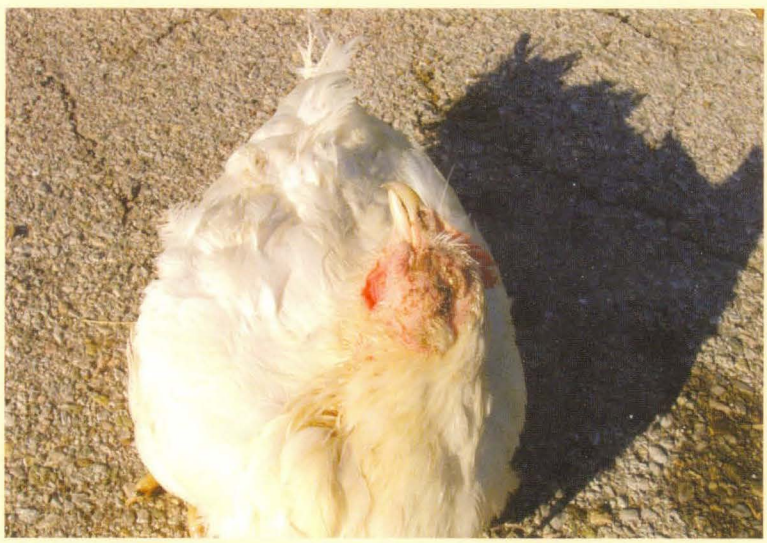




78. The fibrinous caseous exudate accumulated in wattles sometimes leads to gangrene of the covering skin.

79. In turkeys, a common finding is the unilateral or bilateral croupous pleuropneumonia.



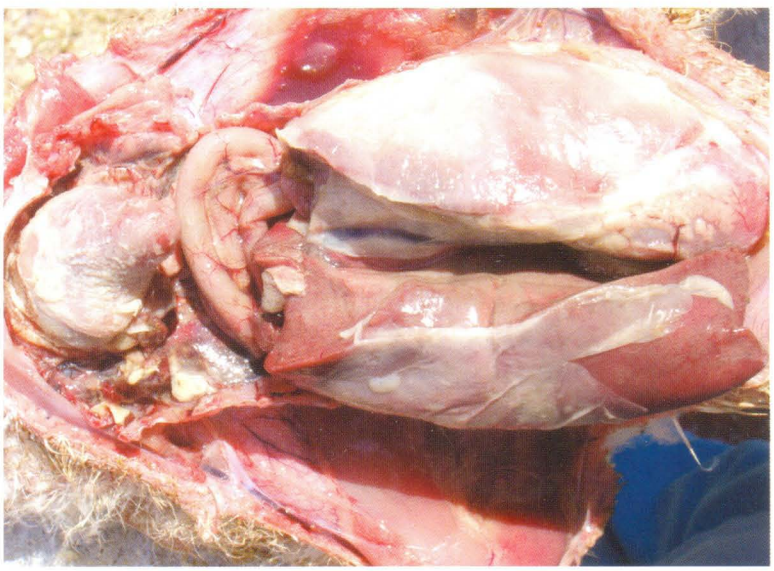


80, 81. The inflammation could possibly be spread from sinuses to adjacent air-filled skull bones with subsequent necrosis and onset of neurological signs (opisthotonus and torticolis). **The diagnosis** is made on the basis of disease history, clinical signs, the lesions and the results of bacteriological studies.

Fowl cholera should be differentiated from acute *E. coli* septicaemia, erysipeloid, fowl typhoid etc. The immunization of birds at the age of 8 - 12 weeks gives very promising results. Many antibiotics and sulfonamides could lower death rate, but at discontinuation of the treatment, the disease could recur. Sulfonamides are appropriate for treatment, but they inhibit egg-laying.

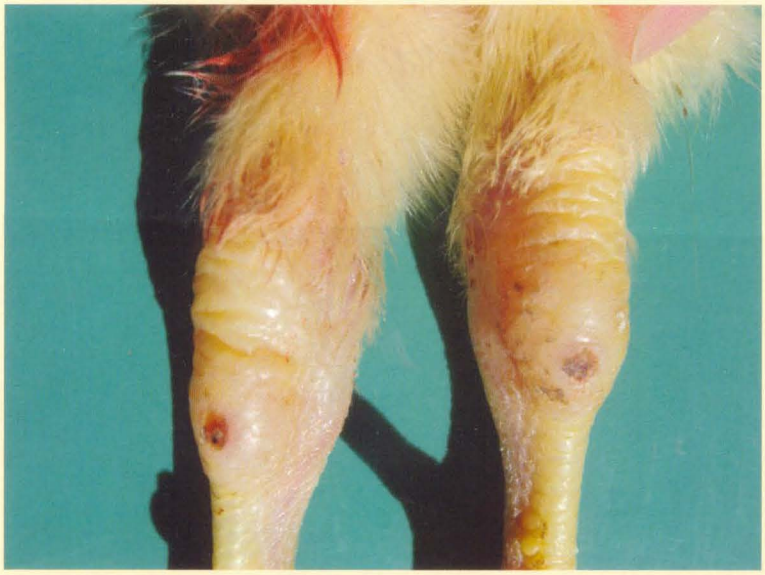
RIEMERELLA ANATIPESTIFER INFECTIONS

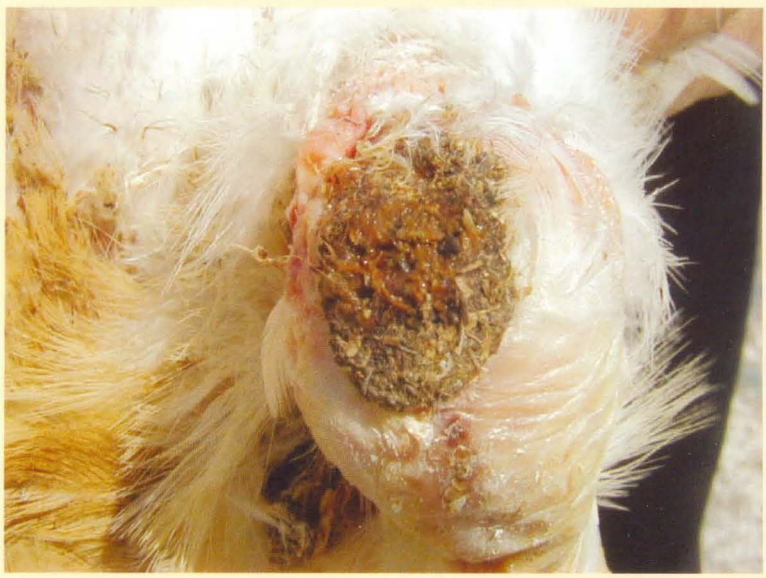
82. *Riemerella anatipestifer (RA)* infection is a contagious disease in domestic ducks, turkeys and other fowl. It is encountered as acute or chronic septicaemia and is characterized with serous fibrinous polyserosites. The respiratory tract could be also affected. The ducklings at the age of 18 weeks are especially susceptible. Clinically, sneezing, cough, trembling of the head and neck, ataxia and greenish diarrhoea could be present.



83. The most characteristic gross lesion is the deposit of fibrinous exudate on the pericardium, the liver capsule or air sacs. The chronic lesions affect the skin and the joints. Although a tentative **diagnosis** could be made on the basis of observed clinical symptoms and lesions, it is confirmed upon the isolation and identification of RA. The RA infection should be distinguished from septicaemiae due to *P. multocida*, *E. coli*, *Salmonella* etc. The treatment with antibiotics (*Flumequine*) and sulfonamides (*Trimetoprim, Sulfadiazine*) has a varying success.

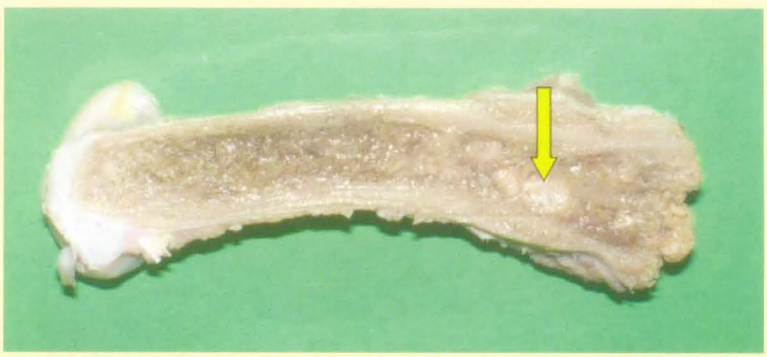
STAPHYLOCOCCAL INFECTIONS

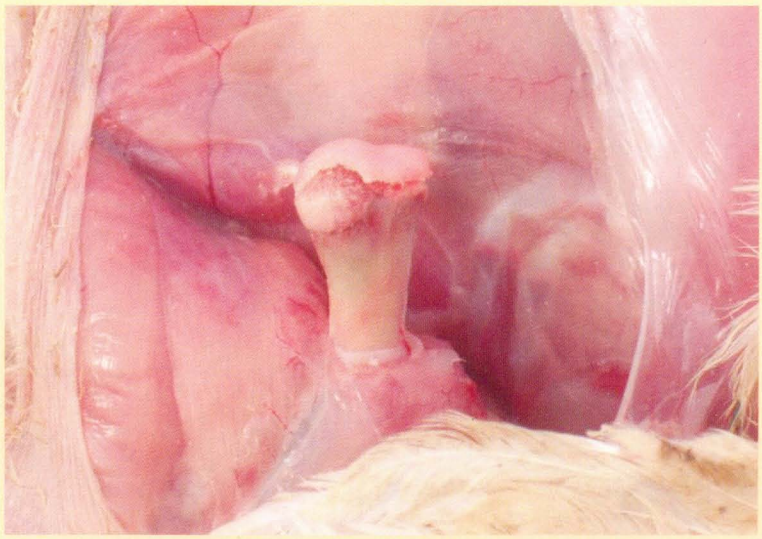


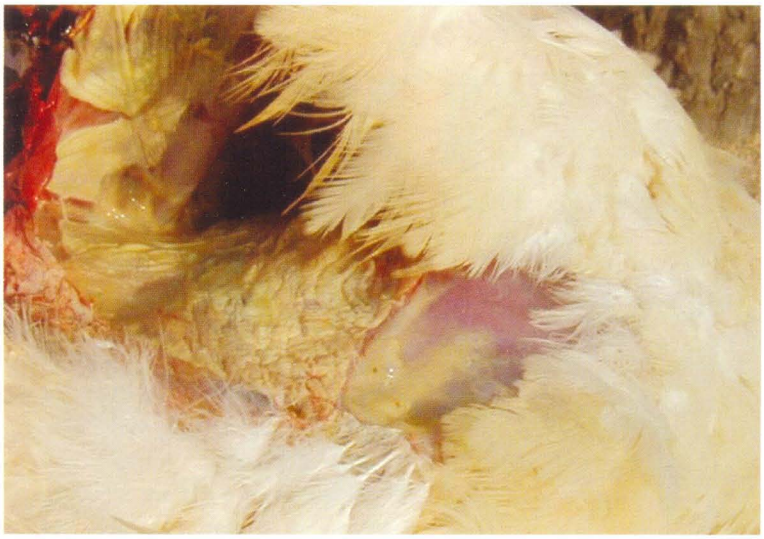


84, 85. *Staphylococcus aureus* infections are commonly seen in poultry. Usually, bones, tendon sheaths and joints, particularly coxofemoral and tibiotarsal joints are affected. The clinical signs include unilateral or bilateral lameness, reluctance to move and lying down. When the tibiotarsal joints are affected, swellings, fever and sometimes necroses of overlying tissues and purulent exudation are observed.

86, 87. As a sequel to septicaemia, osteomyelitis could occur. The lesions are usually detected in the region of the proximal femur, where inflammatory necrotic foci in the bone marrow and partial or complete fracture of the femoral head are observed.

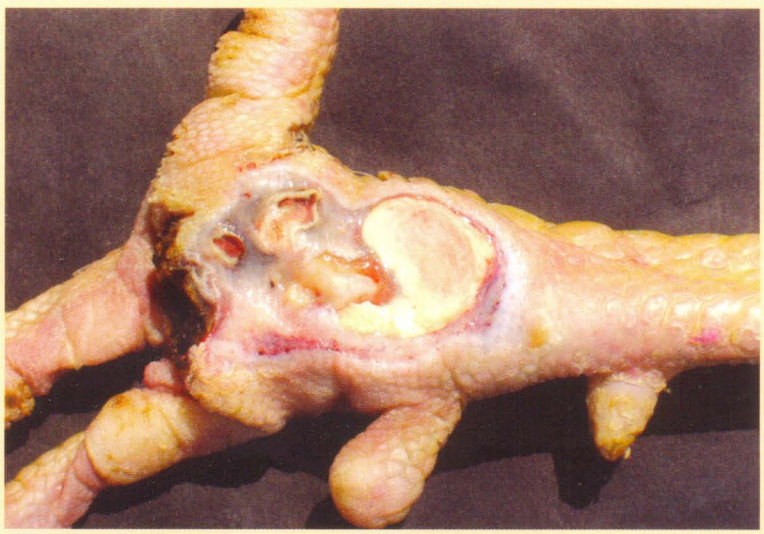


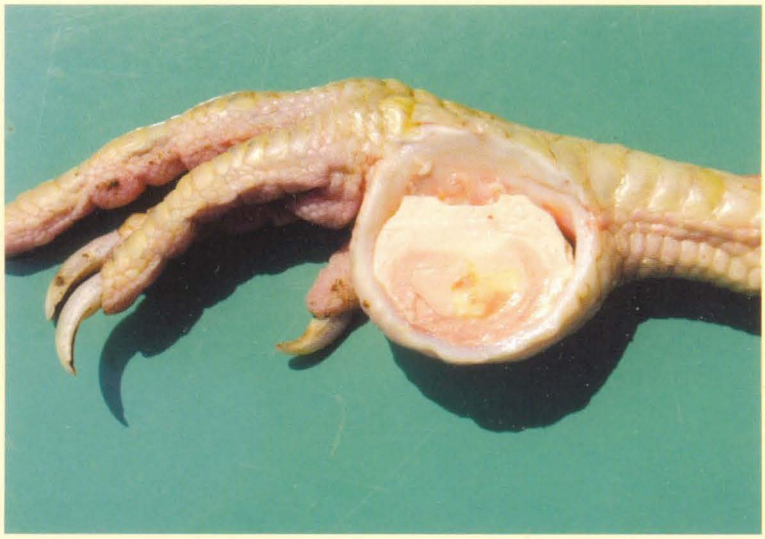


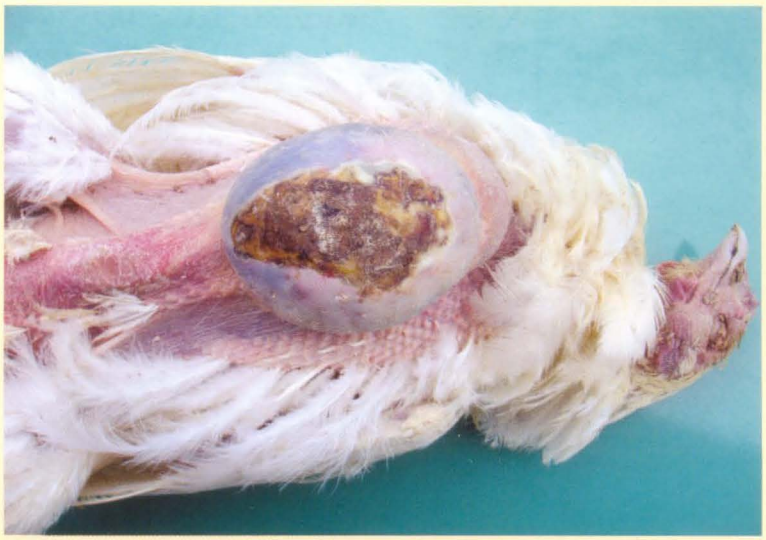


88. The outcomes of skin injuries are the appearance of cellulites, characterized by extensive purulent inflammation of the subcutaneous tissue. Most staphylococcal isolates are identified as *Staphylococcus aureus*, Gram-positive cocci, arranged in clusters.

89, 90. Plantar abscesses are a kind of local purulent inflammation. They are located on the plantar surface or adjacently to it and appear following puncture wounds. Staphylococci are moderately resistant to common disinfectants. Chlorine-containing disinfectants are efficient in the absence of organic matter.



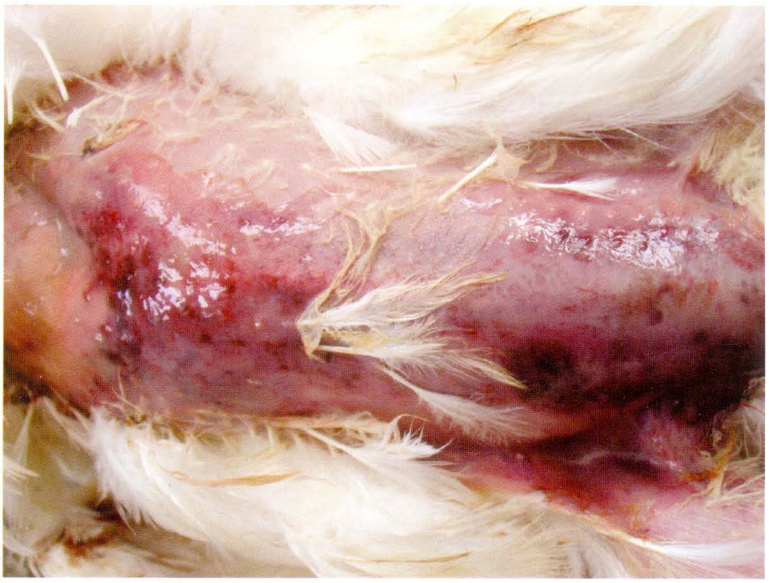


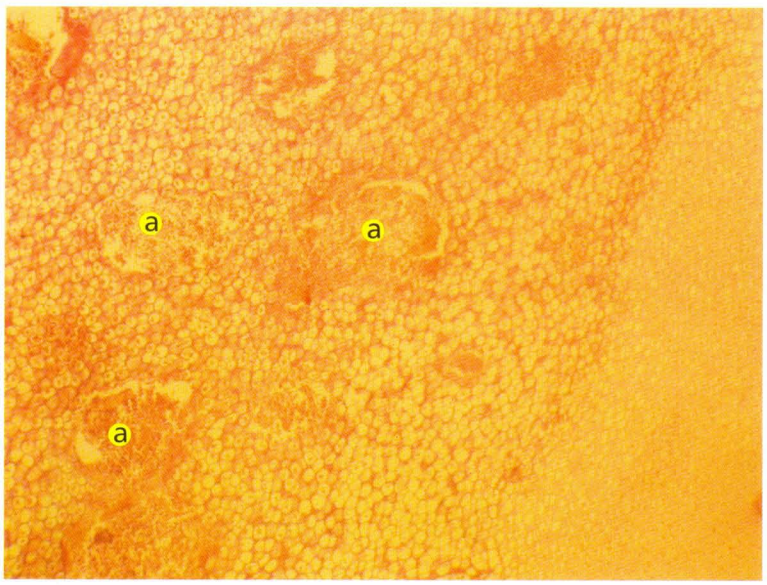


91, 92. Other local forms of staphylococcosis are sternal bursites. The sternal bursa is enlarged at a various extent because of gathering of purulent substance. Sometimes, the covering skin is necrotized. The microorganism is widely distributed in the environment and mainly on the skin. Most of *Staphylococcus aureus*-induced lesions are associated with skin injuries, debeaking, finger cuts. All categories of birds are affected. Toxicogenic strains are able to induce food intoxications.

93. In a septicaemic staphylococcal infection, hyperaemia, enlargement and various-degree coagulation necroses in the liver or the spleen are observed. As staphylococci are ubiquitous, their presence could not be prevented. The measures should be directed toward minimizing the possibilities for traumas of skin, respiratory and intestinal mucosa.

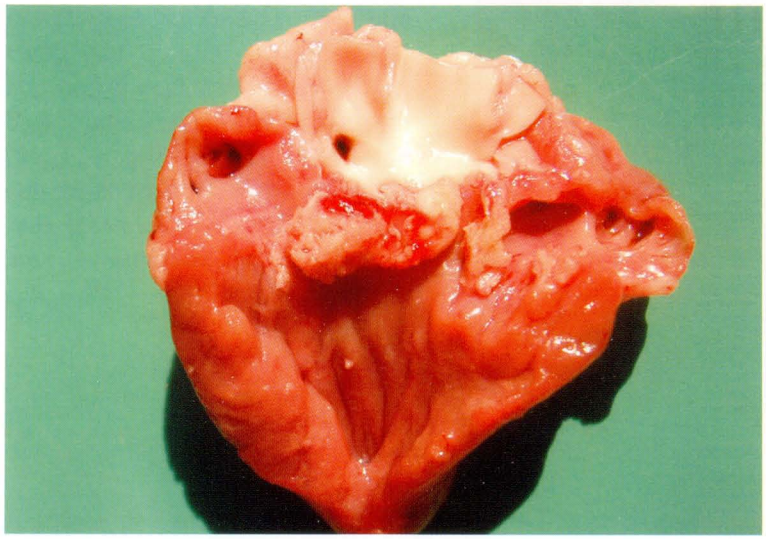
94. A typical secondary infection is staphylococcal gangrenous dermatitis that is usually resulting from immunosuppressive infections (IBD, CIA). Affected skin areas are dark red to blue-greenish, moist and sharply defined from the adjacent healthy skin.





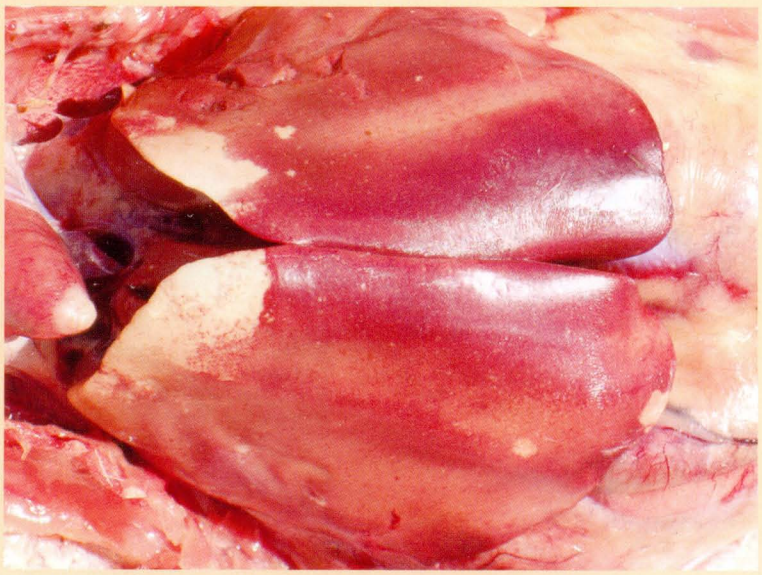
95. Histologically, staphylococcal lesions consist of necroses, bacterial colonization and heterophilic infiltration. The isolation and identification of *Staphylococcus spp.* confirms the diagnosis. From a differential diagnostical point of view, infections with *E. coli, S. gallinarum, P. multocida, M. synoviae* and reoviruses should be considered.

STREPTOCOCCAL INFECTIONS



96. Streptococcosis in poultry is encountered as acute septicaemia or chronic infections with a death rate between 0.5 and 50%. The agent that is most commonly associated with several pathological conditions in poultry is *S. zooepidemicus*. The lesions in chronic streptococcal infections include arthrites, tenosynovites, myocardites and valve endocardites. Endocardites affect predominantly mitral valves and less frequently, aortal and tricuspid valves.

97, 98. Other lesions related to endocardites, are heart, liver and spleen infarctions. Liver infarctions are usually peripherally located on margins, have a pale creamy colour and are sharply demarcated. The penetration of the infection occurs mainly via the oral or aerogenic route, but could also enter through the injured skin, especially in battery cage layer hens. **The differential diagnosis** includes other bacterial septicaemias as staphylococcosis, fowl cholera, *E. coli* infections etc.

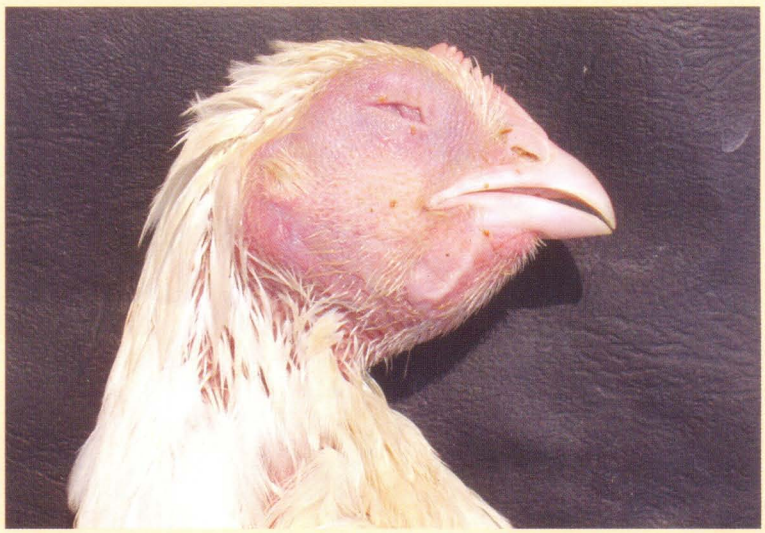


MYCOPLASMOSES

MYCOPLASMA GALLISEPTICUM INFECTIONS

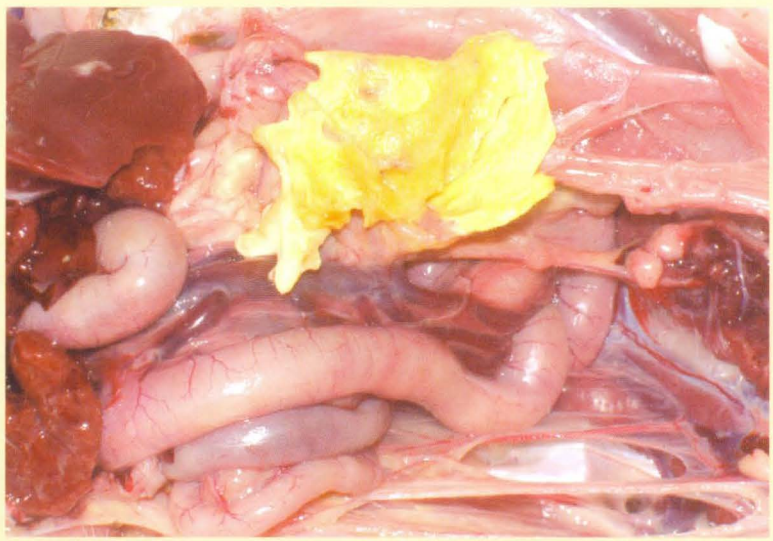
(chronic respiratory disease, respiratory mycoplasmosis, infectious sinusitis in turkeys, MG)

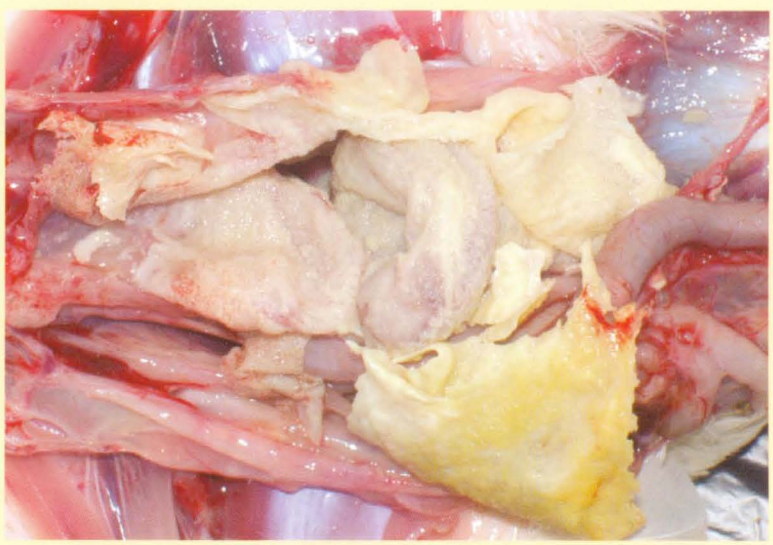
99, 100, 101. MG is characterized by respiratory symptoms and a prolonged course of the disease. Particularly susceptible are hens and turkeys at all ages. The aetiological agent is *M. gallisepticum*. In many cases however, the pathogenicity of the microorganism is enhanced because of its association with any or some of the following agents: *E. coli, P. multocida, H. paragallinarum* and IB or ND viruses. The most characteristic signs in adult flocks are tracheal rales, nasal discharge, coughing, decreased egg production. Most outbreaks are in broiler chickens older than 4 weeks. The course of the disease is more severe during the winter and in cases of associated infections. Often, conjunctivites, facial skin oedema and profuse tear secretion could be observed.

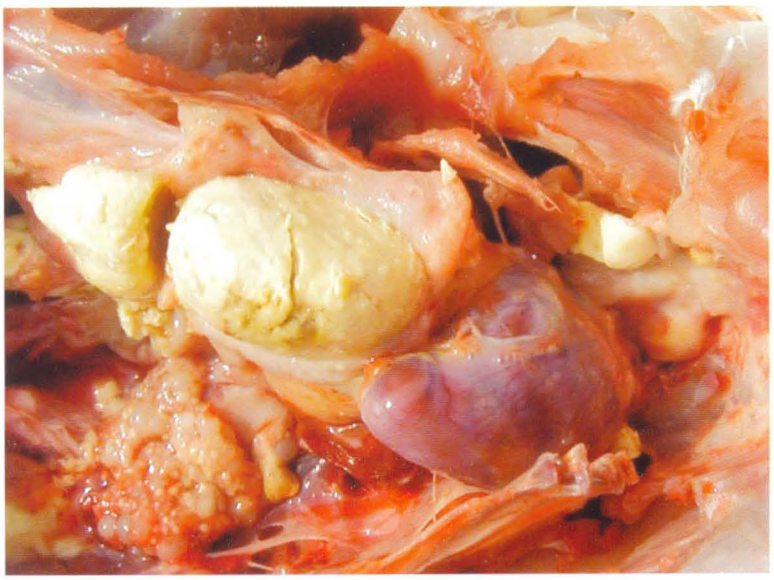


102. In turkeys, unilateral or bilateral swelling of periorbital sinuses, nasal discharge and conjunctivitis are observed. The inflammatory exudate is commonly fibrinous and is detected as diffuse accumulation after removal of overlying skin.

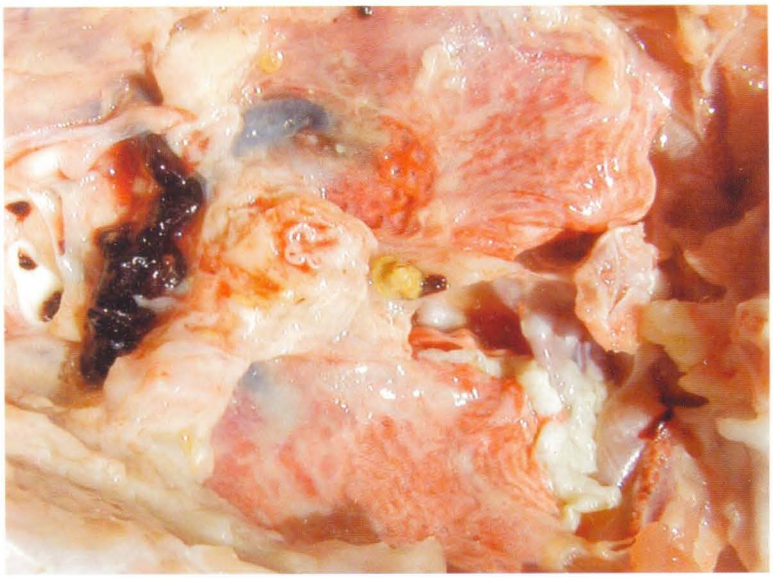
103, 104. The most common gross finding is aerosacculites, the air sacs being filled with fibrinous caseous exudate. The majority of routine chemical disinfectants are effective against *M.gallisepticum* that rarely survives longer than a few days away from the host. The birds could carry the microorganism and be asymptomatic until the disease is triggered by stress factors such as change of the premise, the diet or weather, vaccinations against or infections with IB or ND, increased levels of dust or ammonia.





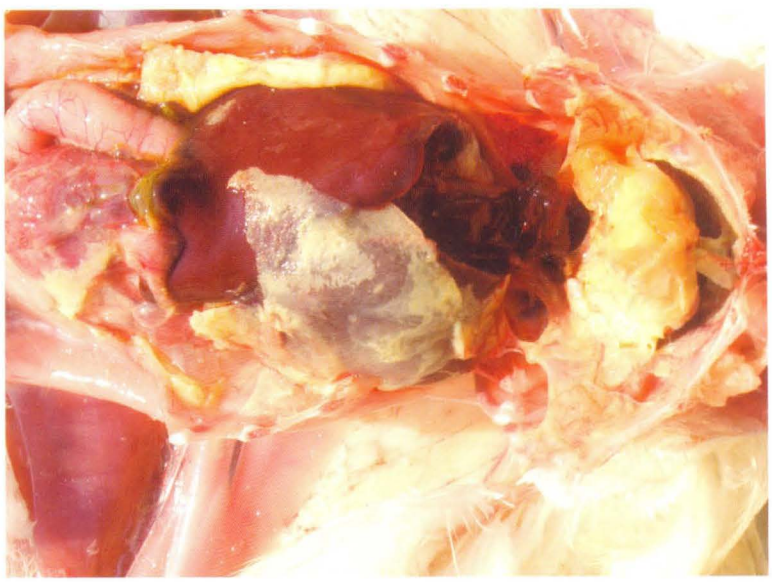


105. In older cases, the content of air sacs is dense and compact. A vertical transmission is done through the eggs of some unapparent carriers. The infected progeny transmits the agent horizontally via airborne route, by coughing or contaminated forage, water and environment.



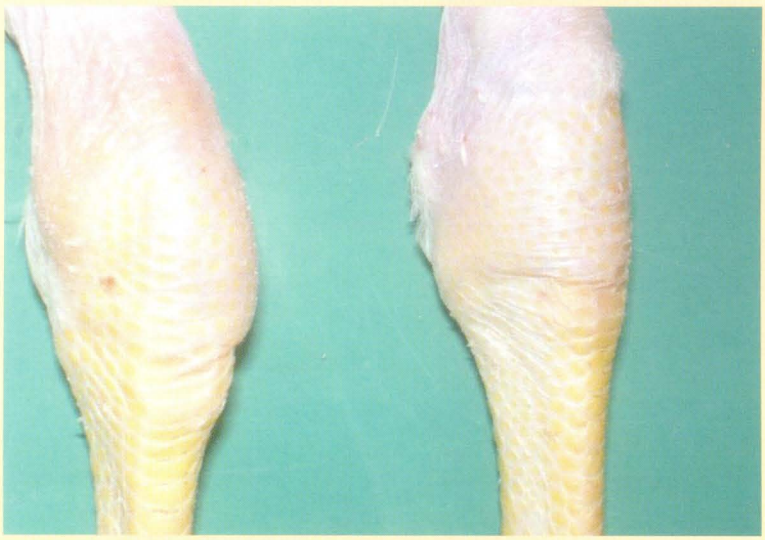
106. Serofibrinous pneumonias, usually bilateral, are a frequent finding.

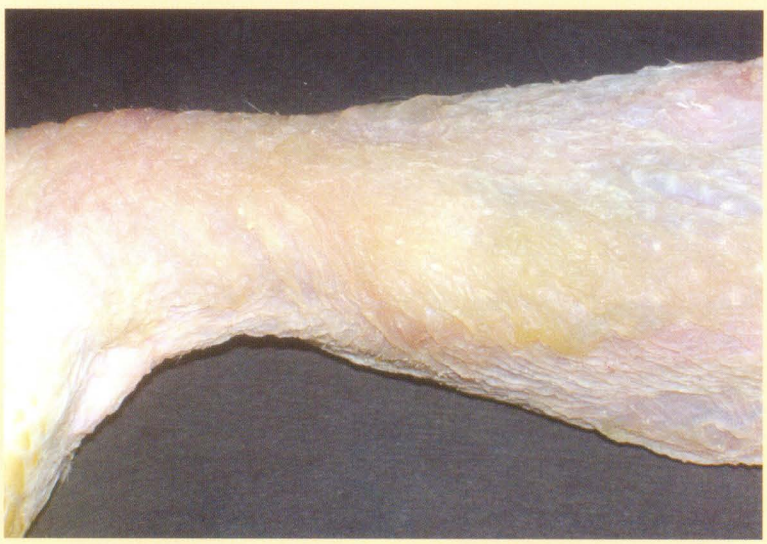
107. Often, the inflammation involves the adjacent serous coats and thus, fibrinous polyserosites occur.



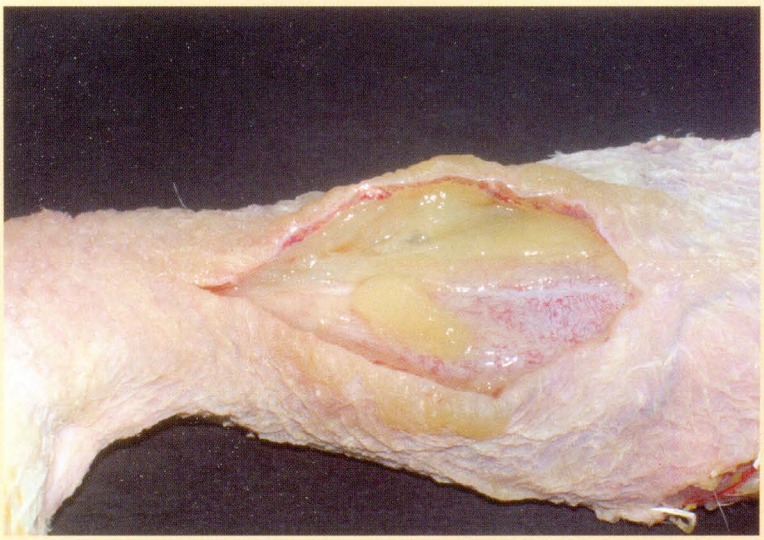
108. Sinusites are relatively rarely observed in hens. The positive agglutination tests of sera in several birds from the flock confirm the diagnosis. MG should be distinguished from other respiratory diseases in poultry. Pulmonary and air sacs lesions could be mistaken with similar findings in *E. coli* septicaemia or aspergillus's. In turkeys, *P. multocida* pneumonia should also be considered.

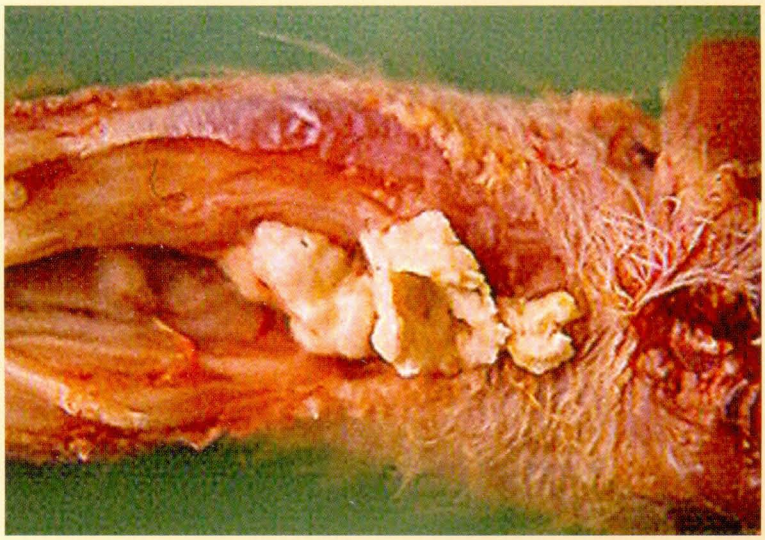
MYCOPLASMA SYNOVIAE INFECTIONS





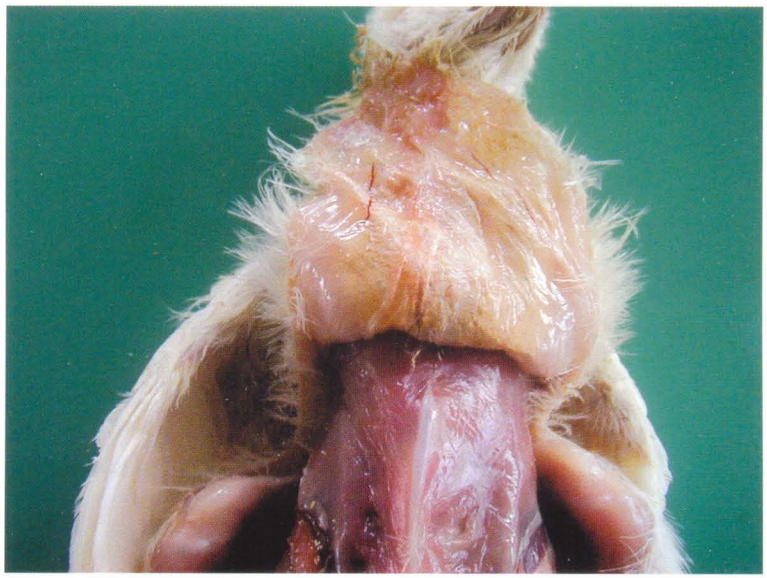
109, 110. *Mycoplasma synoviae* (MS) infections could progress as either acute or a chronic systemic disease with symptoms of arthritis, synovitis and bursitis especially in hens and turkeys. The earliest signs are lameness, lying down and retarded growth. Often, oedemas of tibiotarsal joints and the drumstick are observed. The morbidity and death rates are moderate, under 10%. Young chickens at the age of 4-12 weeks and turkey poults at the age of 10-12 weeks are susceptible. Synovites are encountered all year round, but are prevalent during cold humid seasons or when the litter is wet.



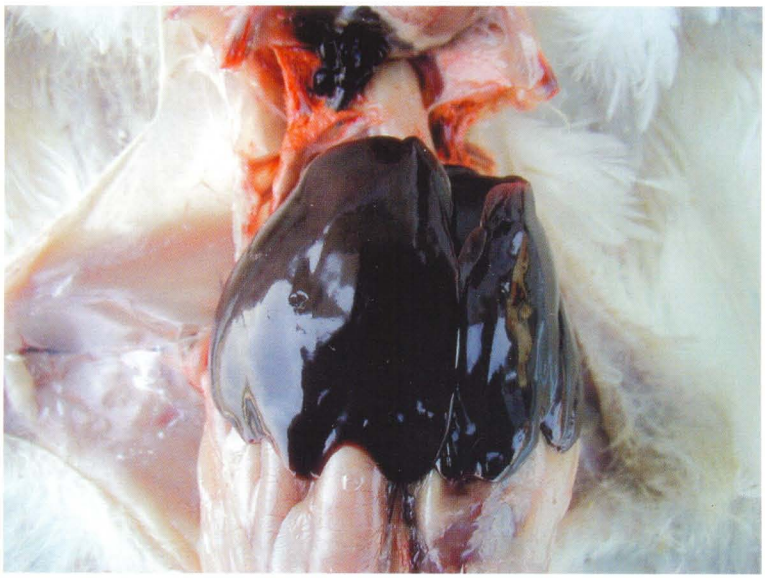


111, 112. Affected birds get progressively exhausted. When the joints and tendon sheaths are open, a serofibrinous exudate is most commonly observed. The aetiological agent is *M. synoviae*. The microorganism shows a certain tropism to synovial structures as joints and tendon sheaths. An important route of dissemination of the agent is the transovarial transmission. The distribution by a horizontal route via the respiratory tract is also possible. The commonly used means of diagnostics is ELISA. MS infections should be differentiated from staphylococcal infections, reoviral arthritis and RGT (see RGT).

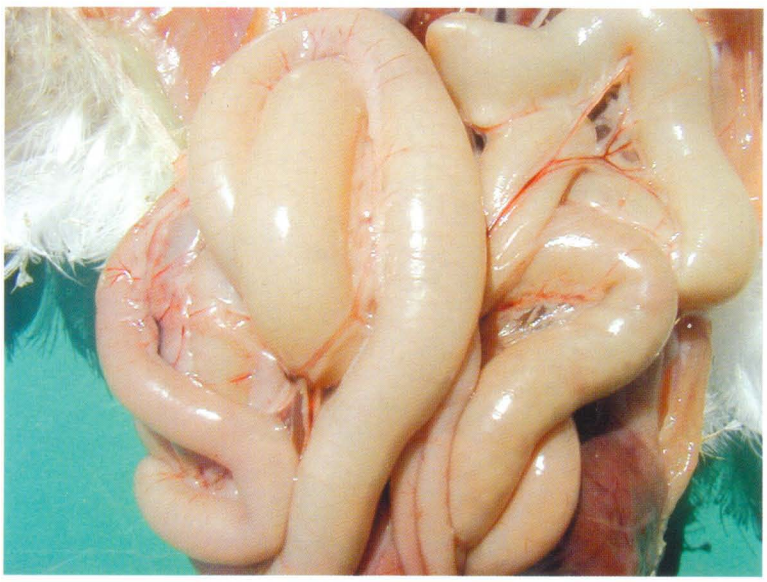
NECROTIC ENTERITIS



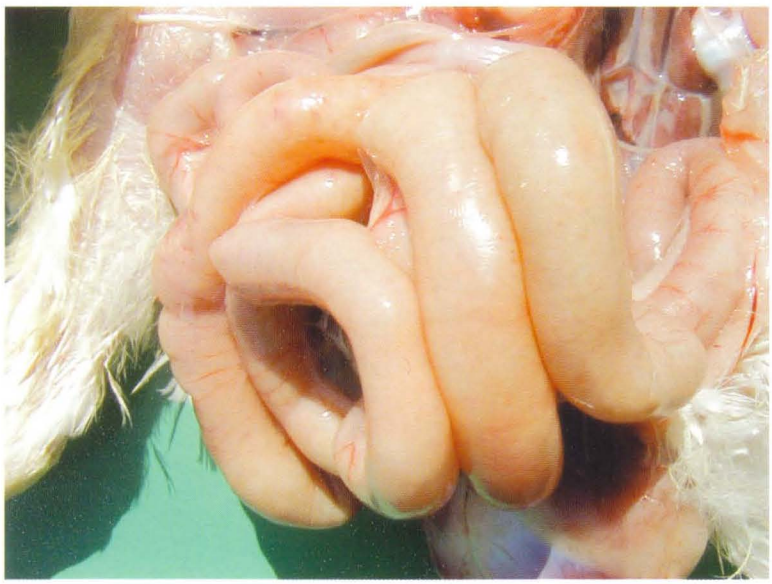
113. Necrotic enteritis (NE) is an acute *Clostridium* infection characterized by severe necroses of intestinal mucosa. The disease begins suddenly, with a sharp increase in death rate. A strong dehydration is observed. The skin is sticked on or adhered to body musculature and is hardly removed.



114. Chickens at the age of 25 weeks are usually affected, NE is also encountered in hens particularly near the period of the beginning of egg laying or peak egg laying, most commonly associated with coccidiosis. In acute cases, marked congestion of liver, responsible for its dark red to black appearance, is present.



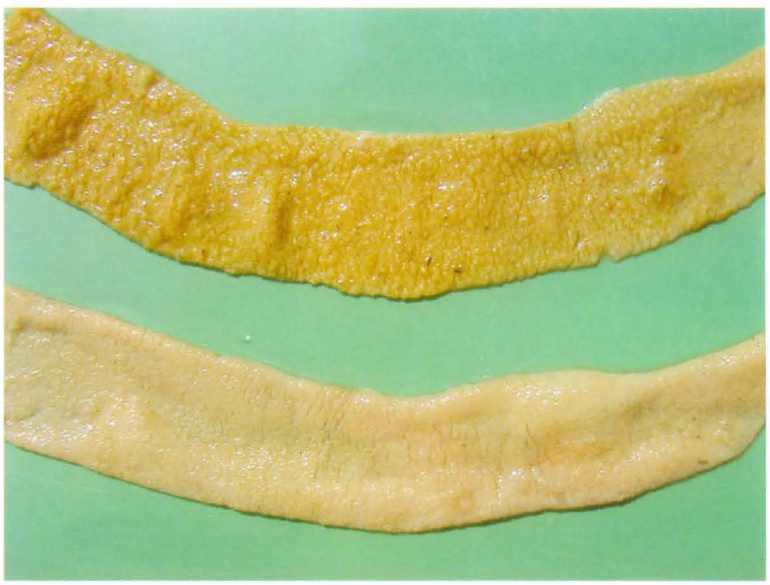
115. The aetiological agent is *Clostridium perfringens*, mainly from type A and more rarely from type C. The produced α and β toxins, from *C. perfringens* type A and type C respectively, are responsible for the necrosis of intestinal mucosa. The small intestine is often distended with gases and the necrotic mucosa is visible through the wall.

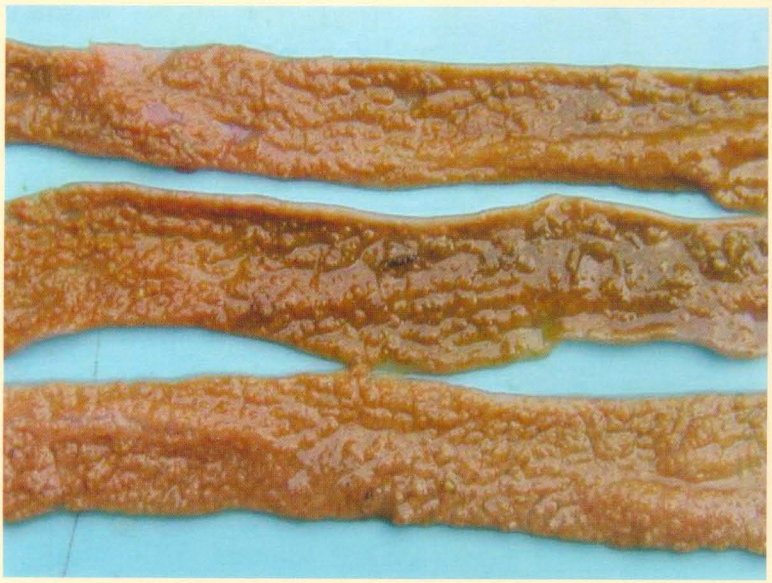


116. *Cl. perfringens* is ubiquitous and normally reside into the intestinal tract. The alterations are particularly in the jejunum and the ileum because of their higher pH and the lower oxygen content in these areas. Sometimes, haemorrhages are seen through the intestinal wall.

117. The intestinal lumen is filled with brownish watery content, mixed with gas bubbles.

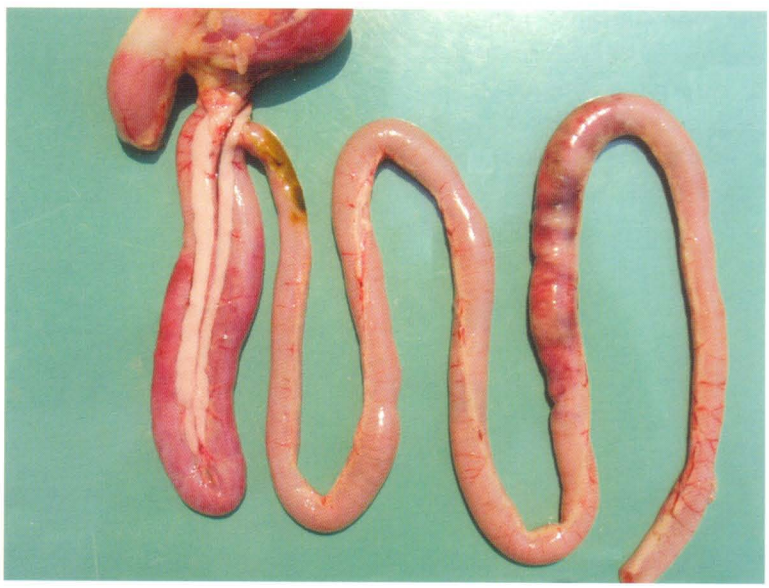
118. The necrotic mucosa acquires a greyish-creamy or greenish appearance. Sometimes the mucosa has a flannelette blanket-like appearance.

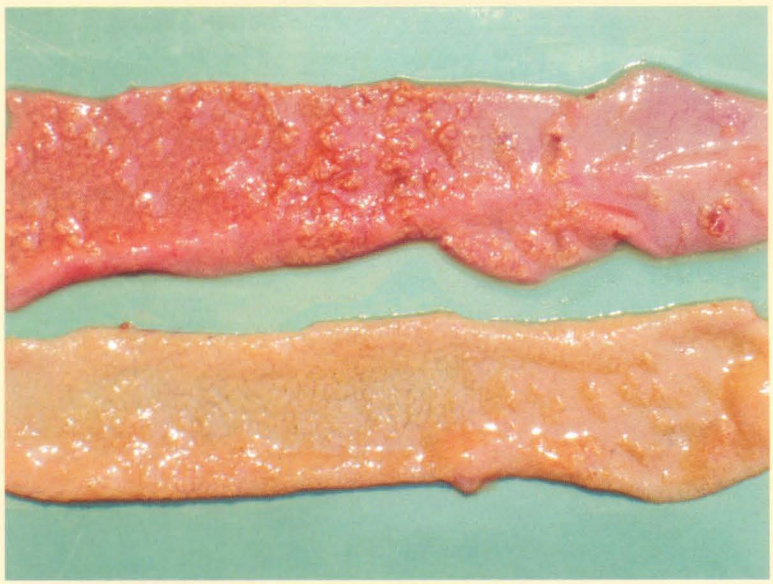


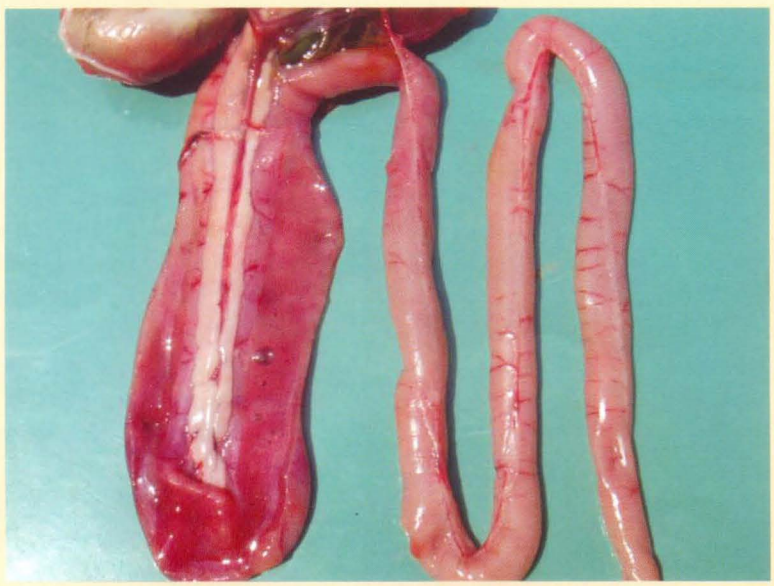


119, 120. In some cases, the mucosa has a linear pattern similar to the bark of a tree. The predisposing factors are injuries of intestinal mucosa by various *Eimeria* species, migration of ascarids, immunodeficiency states due to CIA, IBD, MD, high content of wheat or fish meal in the diet.

121. In cases when NE is associated with small intestinal coccidioses, multiple petechial haemorrhages could be perceived through the wall in different areas along the small intestine.



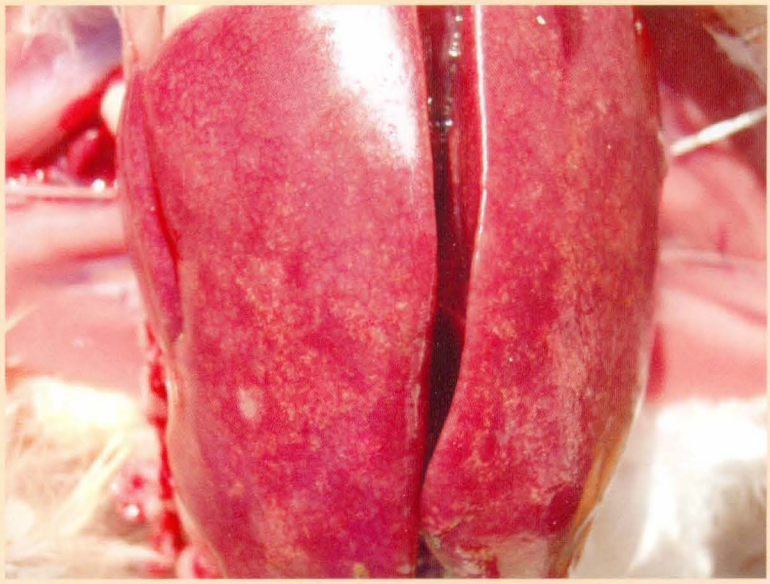


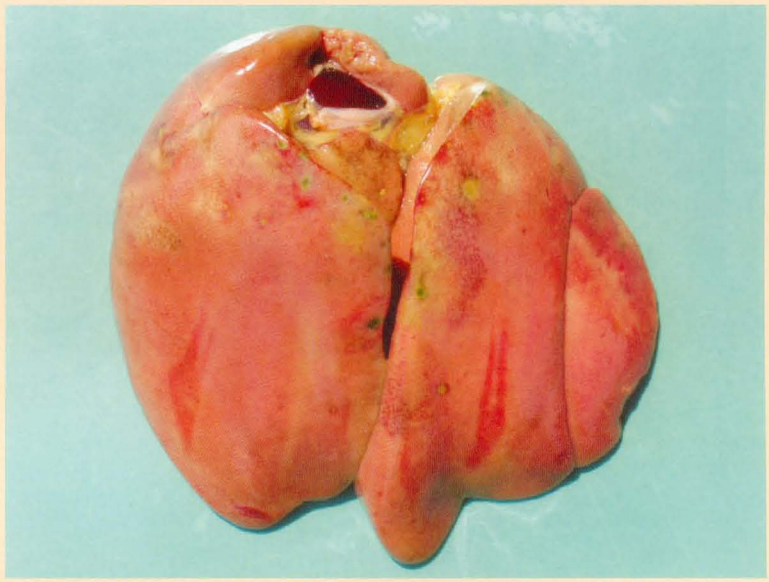


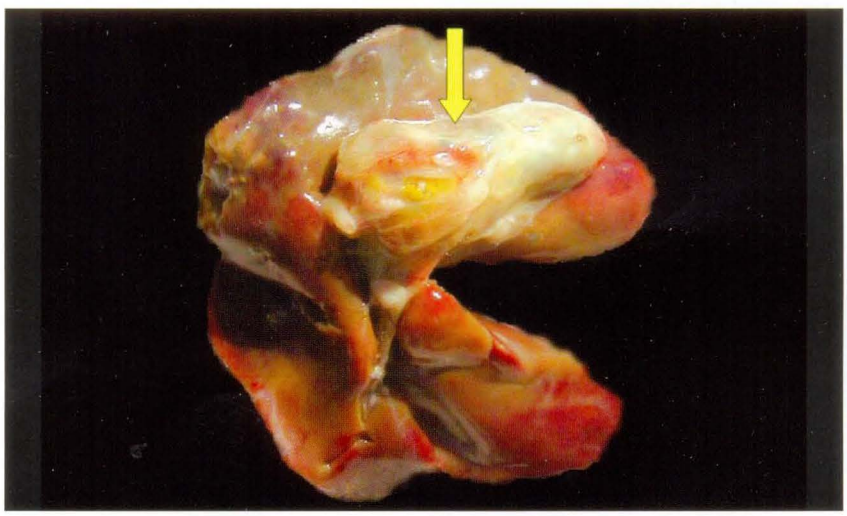
122, 123, 124.
Throughout the simultaneous occurrence of NE and coccidioses, the content of the lumen is bloody, mixed with necrotic detritus and gas bubbles. The **diagnosis** is based on the distinctive gross lesions. When necessary, a histological investigation is performed or attempts for isolation of the causative agent. NE should be distinguished from ulcerative enteritis and some small intestinal coccidioses. The **control** should be aimed at predisposing factors. An appropriate medication of feeds is recommended. A good effect is obtained with oxytetracycline dihydrate (OTC 50% premix). NE could be effectively treated with doxycycline hydrochloride, amoxicillin etc.

CHOLANGIOHEPATITIS IN BROILER CHICKENS

125, 126. Cholangiohepatitis (CAH) in broiler chickens is characterized by inflammatory proliferative and dystrophic necrobiotic alterations in bile ducts and the liver parenchyma. Usually, no clinical signs are observed. The increased daily mortality is insignificant, although in some chickens, a retarded growth and dehydration could be present.
Pathoanatomically, the liver is enlarged and with paler yellow colour. In some cases, its surface has a characteristic acinous appearance and in others is mottled with multiple small greyish-white or greenish foci.

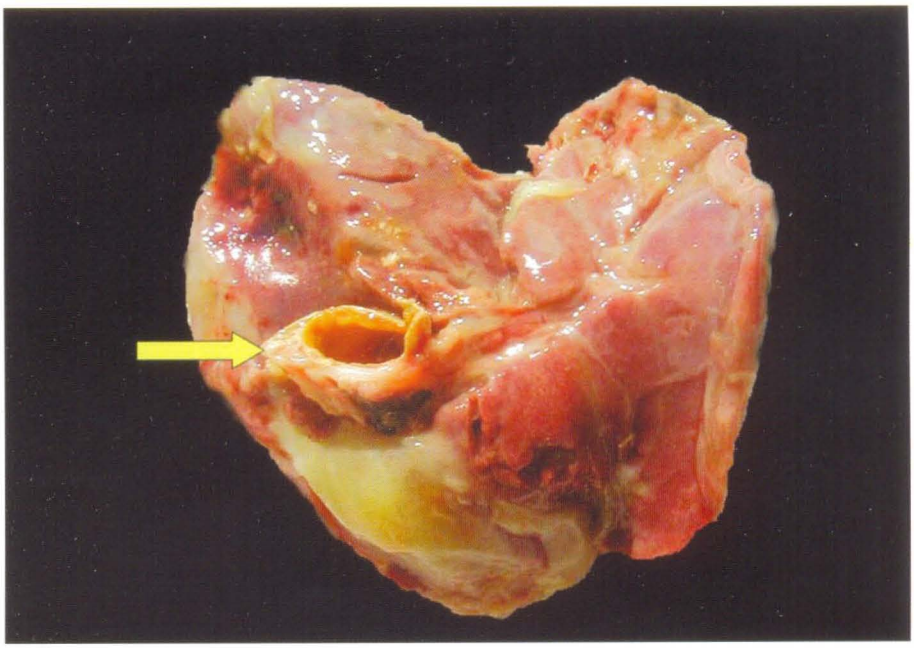


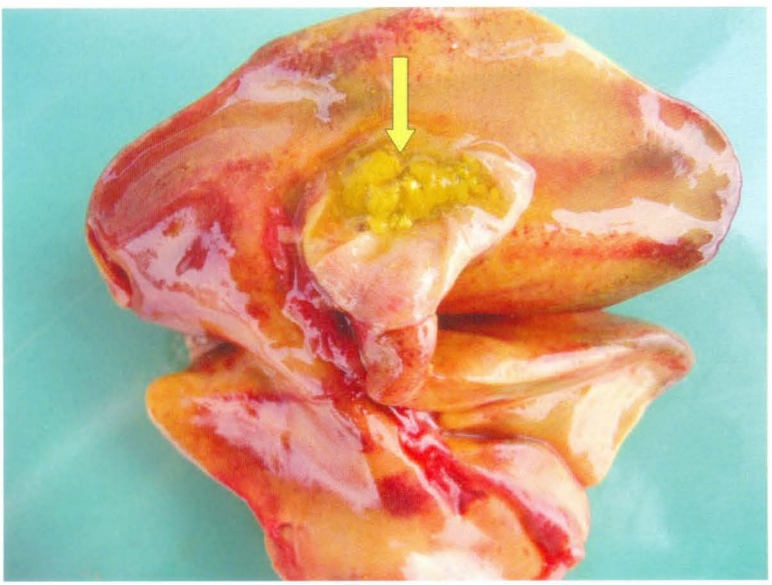




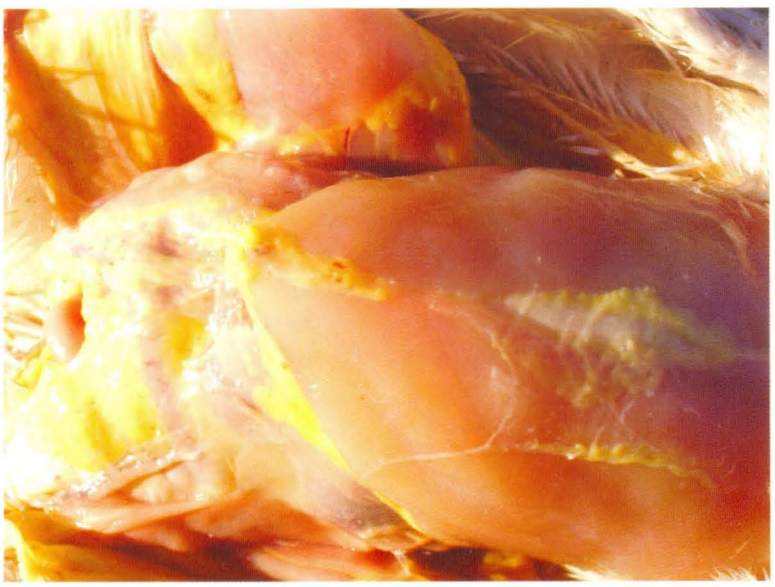
127. The walls of the gall bladder are thickened, sometimes up to 5-6 cm, and opaque. The state is detected in the last phase of the fattening period or in the slaughterhouse. It is possible to observe CHA as an independent disease or associated with necrotic enteritis.

128. A transverse cross section through the thickened gall bladder wall (arrow).



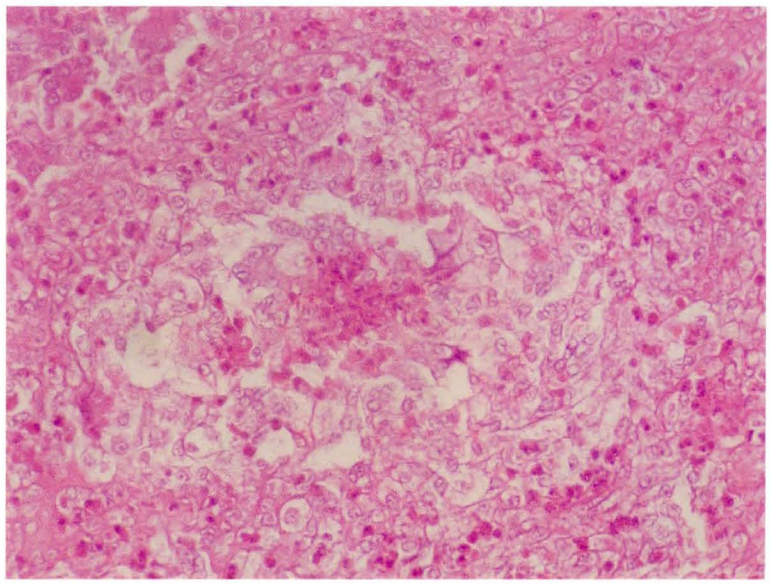


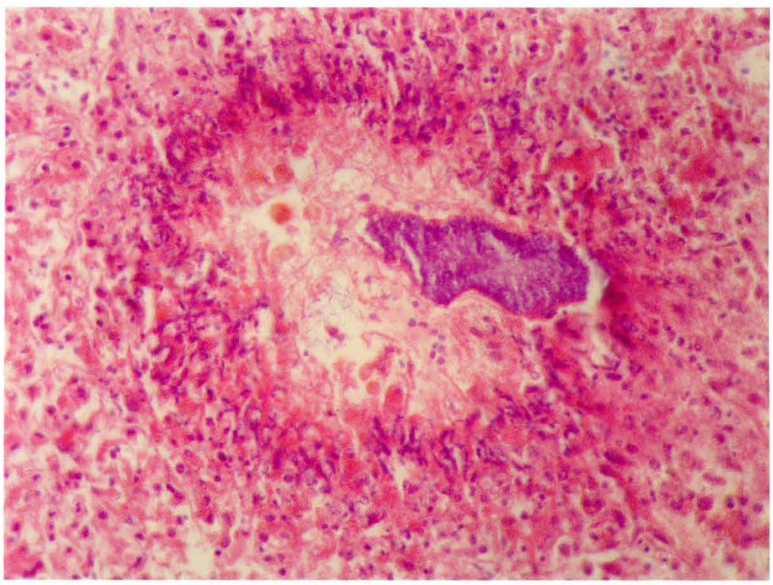
129. *Clostridium perfringens* is the aetiological agent. CAH is experimentally reproduced in broilers by ligation of bile ducts and inoculation with *Cl. perfringens*. The gall bladder is filled with a thick bile secretion or a dense matter with a creamy colour.



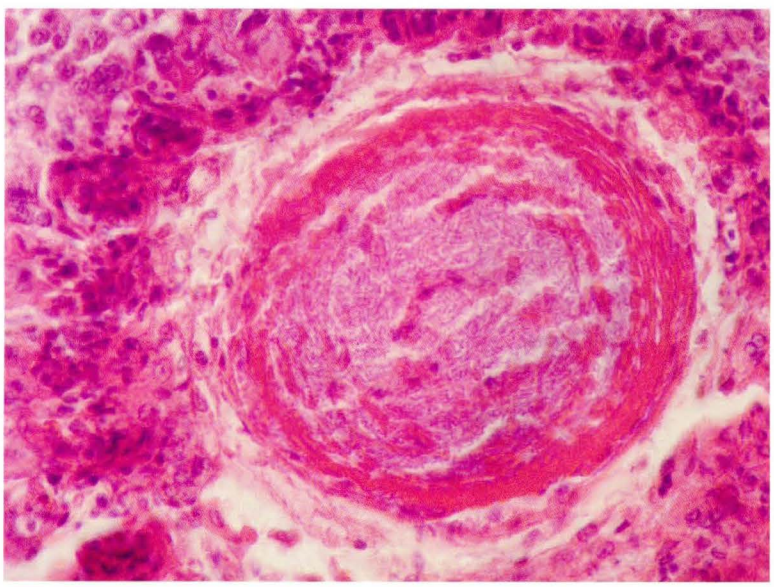
130. In some chickens, the subcutaneous fat and the body fat have an icteric tint.

131. Histologically, the liver lesions are detected at a various stage of development. In the majority of cases, proliferative changes in bile ducts are observed. The overgrown bile ducts form granulomatous structures, surrounded by fine reticular fibres. Centrally, in some granulomas, either initial or advanced degree of necrosis and weak to moderate granulocytic infiltration are observed.



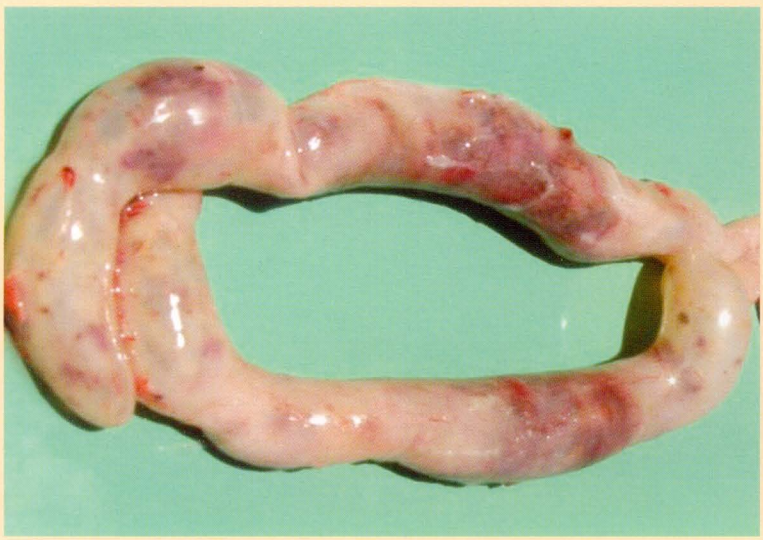


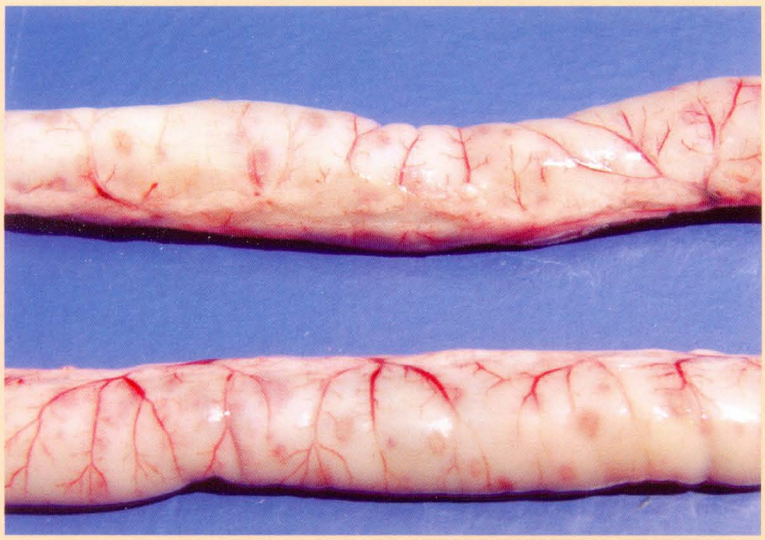
132. In many bile ducts, a biliary stasis is present and within some, among the stagnated secretion, a huge amount of microorganisms are detected. Pericanalicularly, coagulation necroses are frequently noticed. Among some of these foci, hyalinization of the necrotic masses and single microorganisms are present. Around the necroses, a belt of macrophages, lymphocytes and granulocytes is formed.



133. Many Gram-positive bacteria are detected among granulomas, in bile ducts' lumen and into the gall bladder, often accompanied by inflammatory lesions. The mucosa of bile ducts and the gall bladder is frequently necrotized and the wall is thickened because of connective tissue growth. The **diagnosis** is based on the characteristic gross and microscopic lesions. The prevention is similar to that in NE.

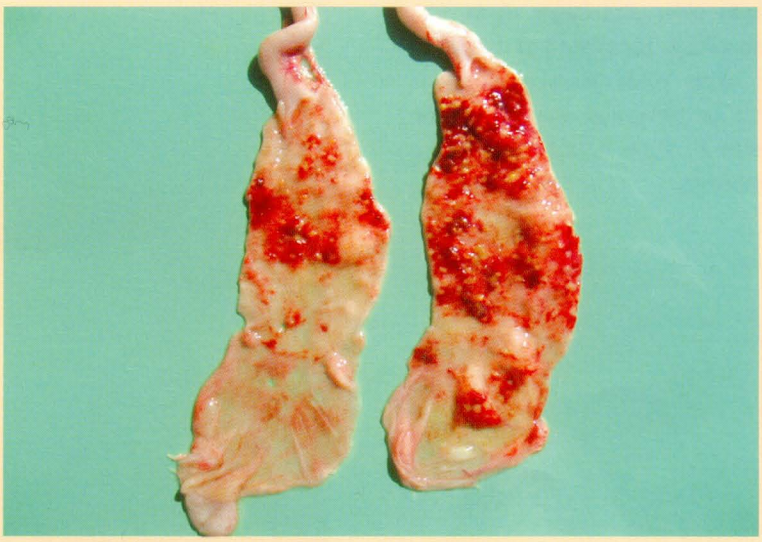
ULCERATIVE ENTERITIS

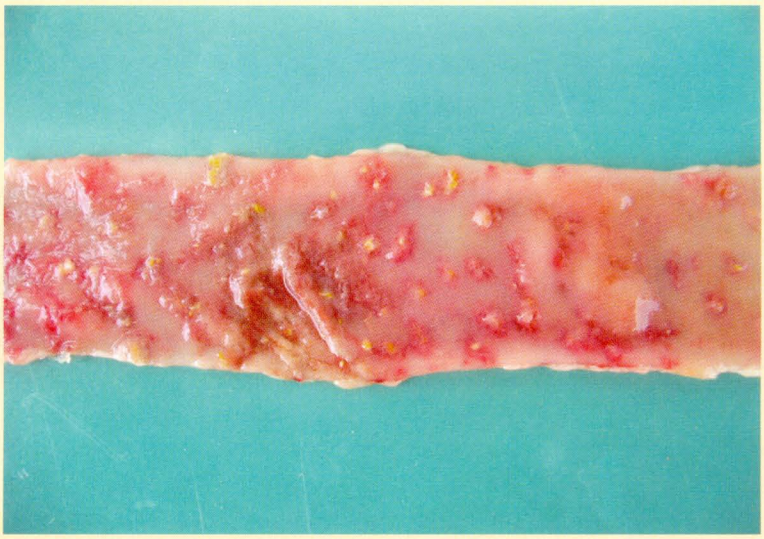




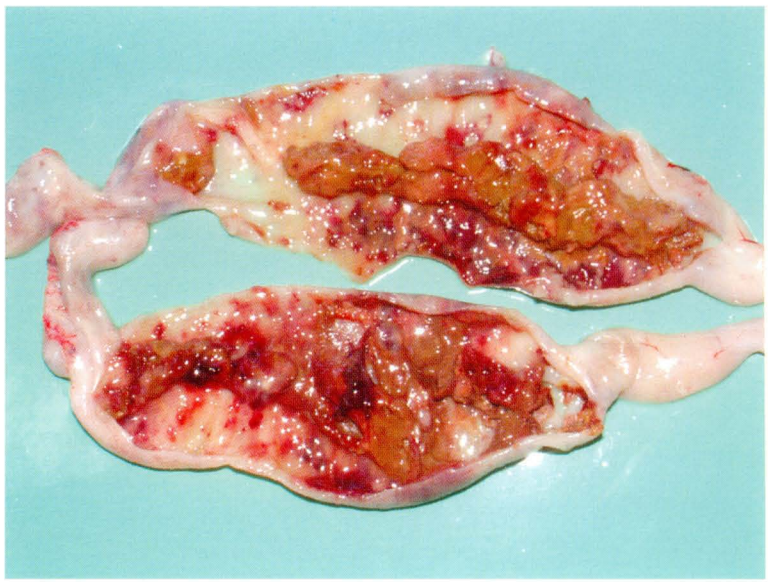
134, 135.
Ulcerative enteritis (UE) is characterized by inflammatory ulcerative and necrotic changes in intestinal mucosa and dystrophic necrobiotic lesions of the liver and the spleen. The clinical signs include a general malaise, ruffled feathers, diarrhoea and anaemia. In many instances, the disease begins with a sudden death. Pathoanatomically, deep button-like ulcers are observed, mainly in caeca and less frequently, in some parts of the small intestine, usually visible through the wall.

136, 137. UE is a problem in all world regions with extensive poultry breeding. Young birds are infected more frequently although the disease is also common among adult quails. The early lesions appear like yellowish foci with haemorrhagic boundaries that could be seen from both the serous and the mucosal surfaces.

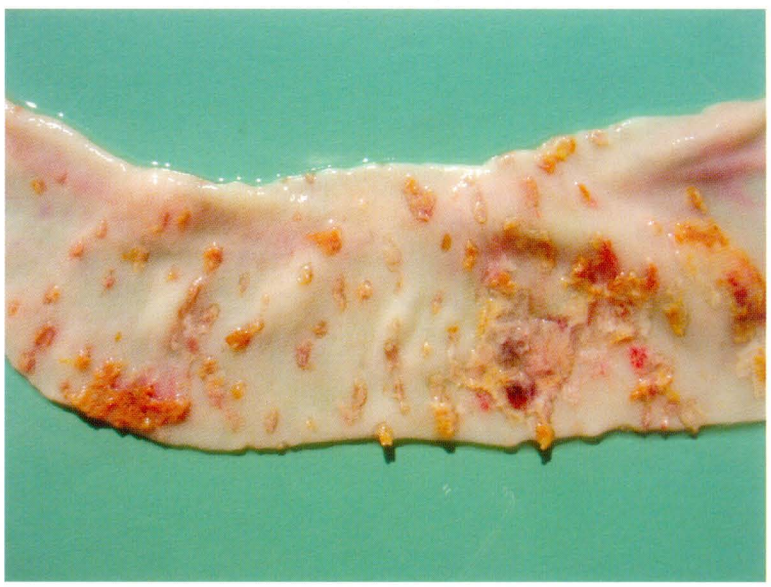


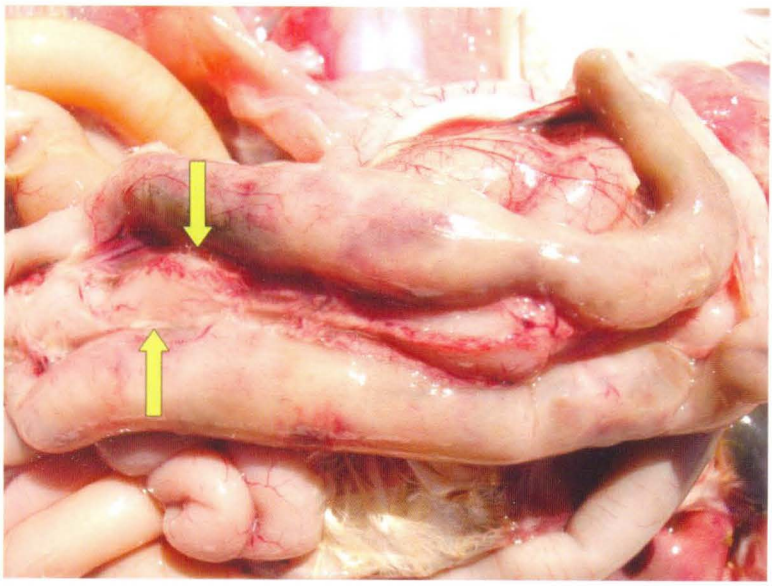


138. The aetiological agent is *Clostridium colinum*, a spore-forming organism, highly resistant to chemical agents and physical alterations. The intestinal content is often mixed with blood.



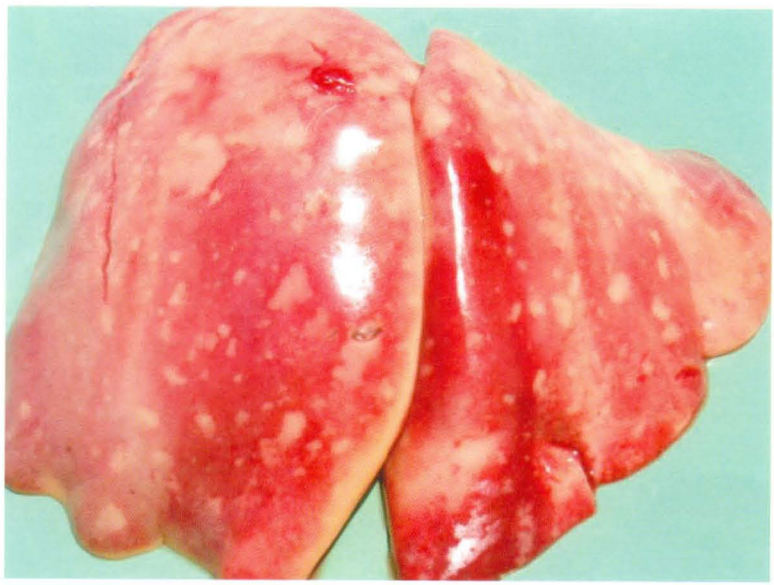
139. In older and larger ulcers, the haemorrhagic zones tend to disappear. The ulcers could have an irregular round or elongated shape and are covered by large necrotic diphtheritic membranes.



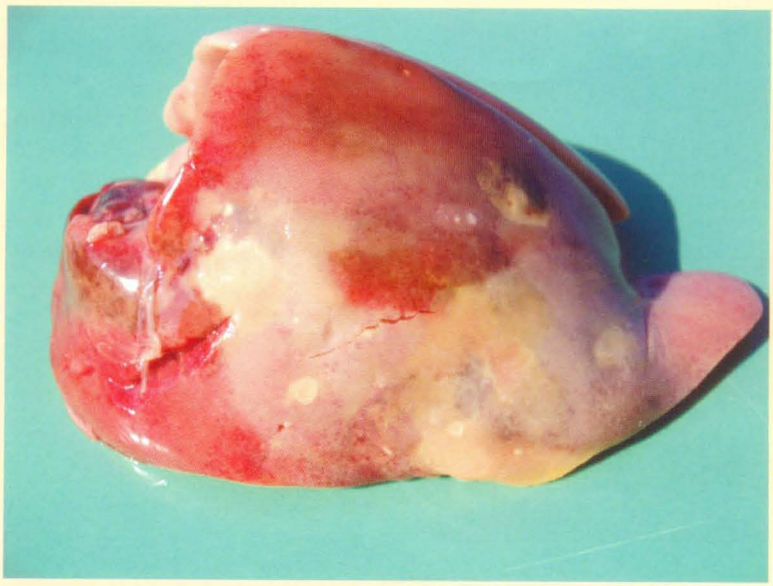


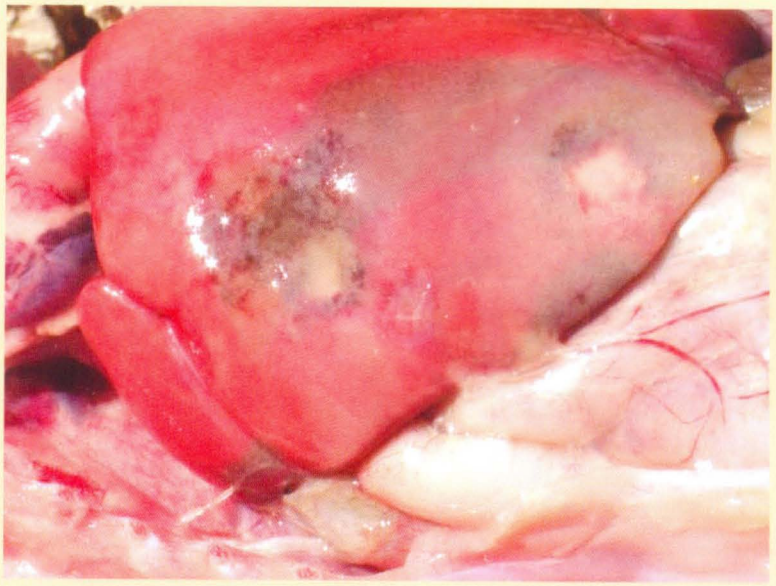
140. Frequently, adhesive peritonitis due to inflammatory involvement of adjacent serous coats is observed. Numerous domestic and wild birds (chickens, quails, turkeys, rock partridges, geese, partridges etc.) are susceptible. The chickens and the quails are the most vulnerable between 4 and 12 weeks of age where as turkeys between 3 and 8 weeks of age.

141. In the liver, a variety of dystrophic changes and necroses with different size and shape are detected. Necrotic foci in some cases are milliary.



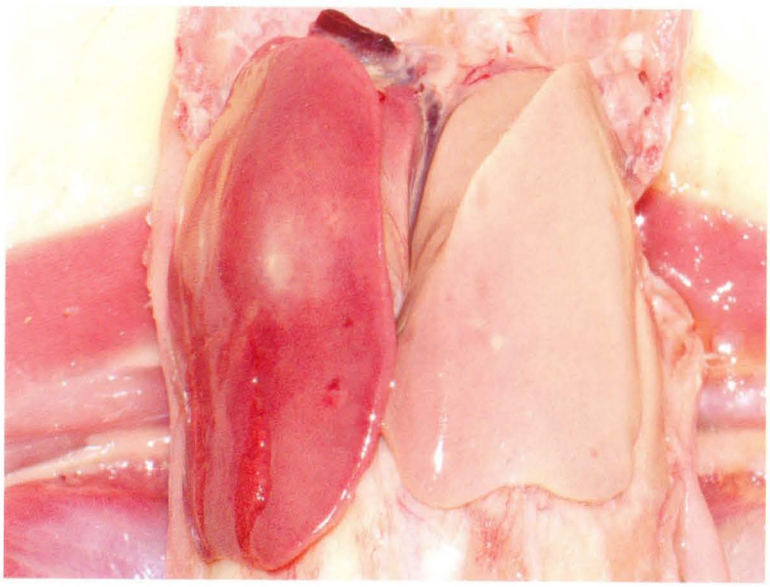
142. Sometimes, liver necroses reach 1 - 2 cm. in diameter and are surrounded by a haemorrhagic zone.



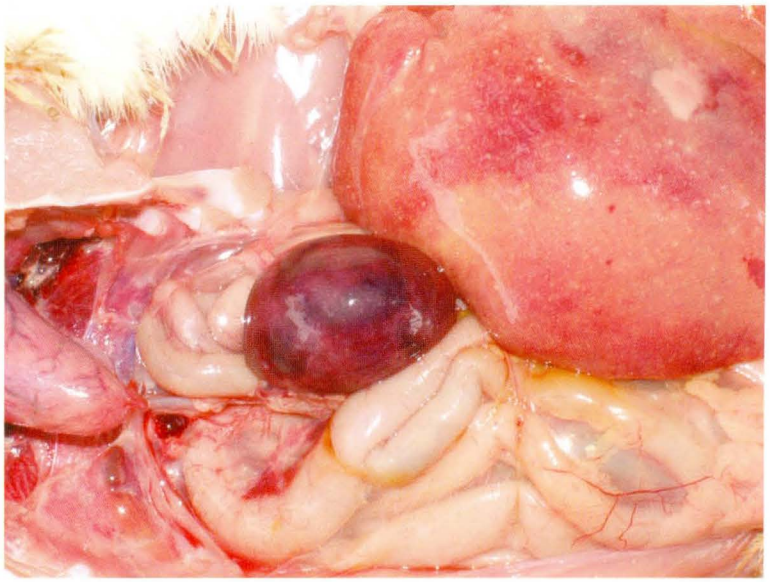


143, 144. In some instances, necrotic foci involve large parts of the liver and are infiltrated by haemorrhages. The aetiological agent is distributed with the excreta of acutely ill and recovered birds and persists in soil for many months. The incubation period is 1 - 3 days. The death rate in chickens varies from 2% to 10% and in quails reaches 100%. The outbreaks of UE in chickens are often associated with or come after coccidioses, CIA, IBD or stress conditions.

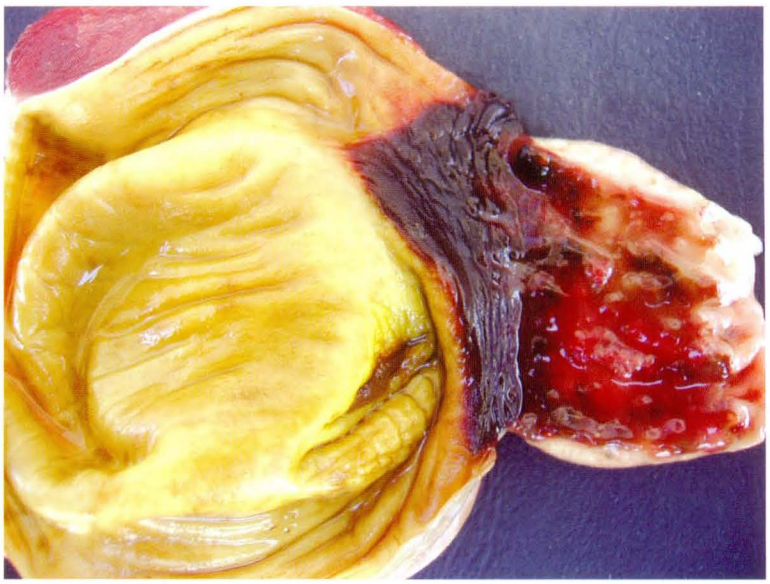
145. Most commonly, necroses are distinguished on the background of a marked parenchymatous dystrophy, affecting partially or totally (uni- or bilaterally) the liver.



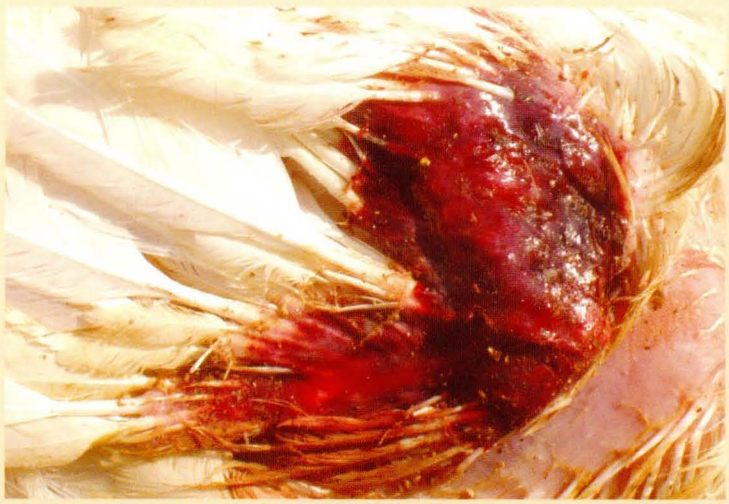
146. The spleen could be enlarged, hyperaemic, haemorrhagic and sometimes, with necroses. **The diagnosis** is based on the typical gross lesions. When needed, imprint preparations are made, a histological study is performed or attempts for isolation and identification of the aetiological agent are made. UE should be differentiated from NE, coccidiosis and histomonosis (typhlohepatitis).



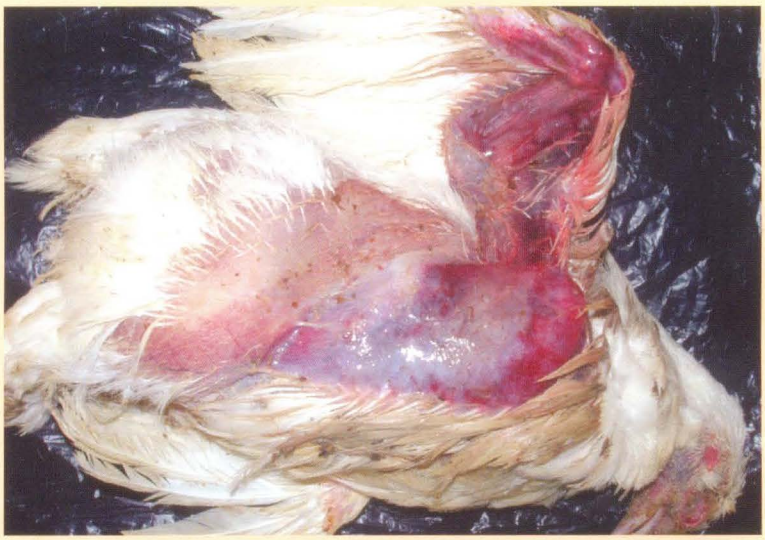
147. In some cases, haemorrhages with various intensities are detected in the mucous coat of the gizzard. **Prevention** - separate housing of the different age groups of birds, avoiding the contact with other avian species. The premedication of forages with some antibiotics and their rotation would prevent the reproduction of Cl. colinum. A good effect is achieved with oxytetracycline dihydrate (OTC 50% premix). UE could be effectively treated with doxycycline hydrochloride, amoxycillin etc.

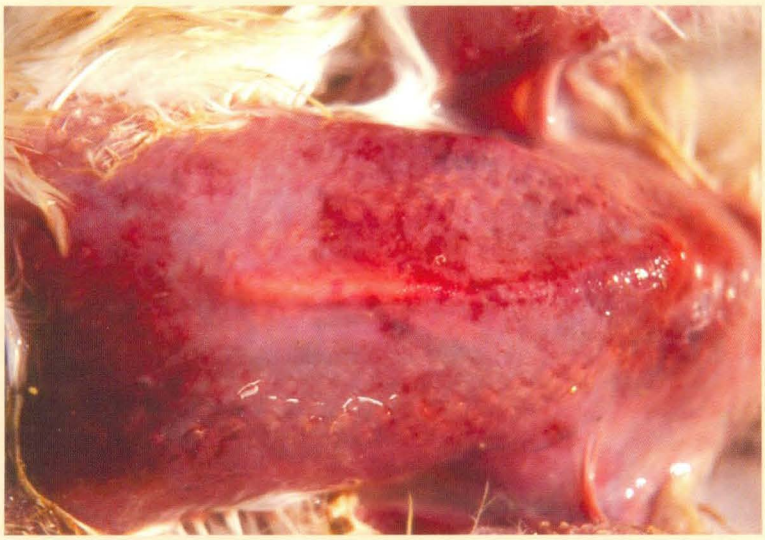


GANGRENOUS DERMATITIS

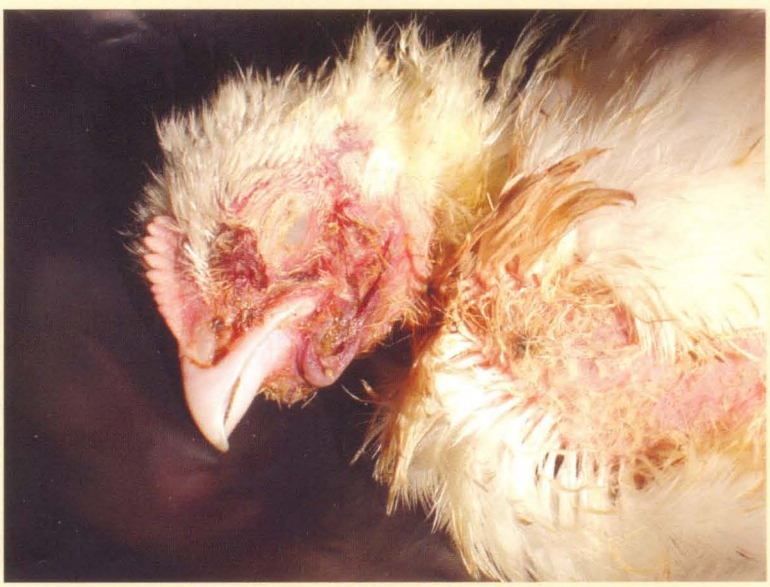


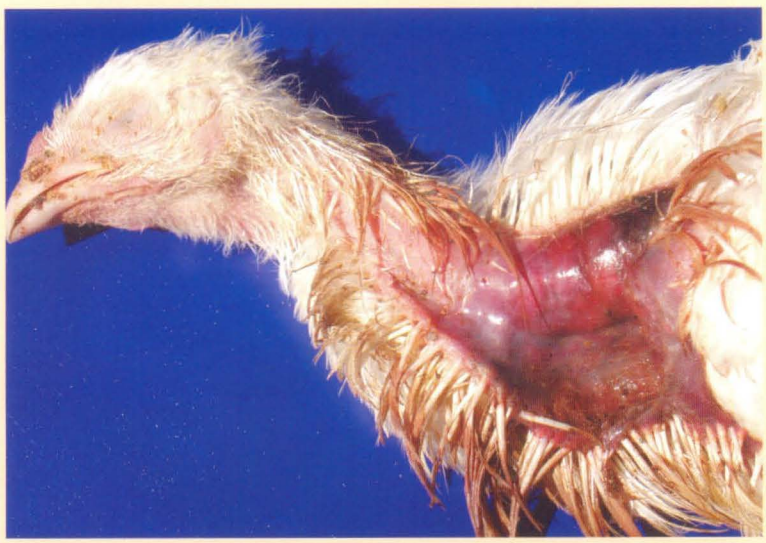
148, 149, 150. The gangrenous dermatitis (GD) is a disease affecting fattening and growing birds, characterized with necrotization of different skin areas and a severe cellulitis of the subcutaneous tissue. The sudden and quick increase in death rates is often the first signal for the incidence of GD. The affected birds die after less than 24 h. The death rate is from 1% to 60%. The lesions are dark red to blue green macerated skin areas, usually featherless, beginning generally from wings and the adjacent areas.

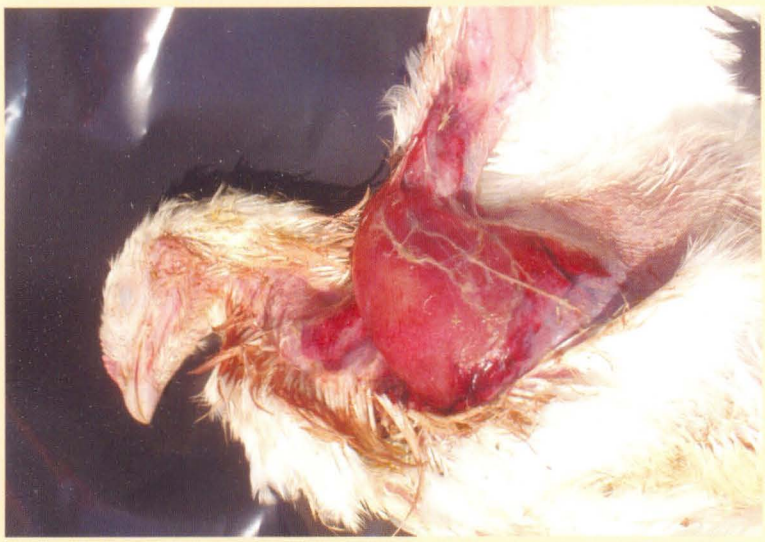




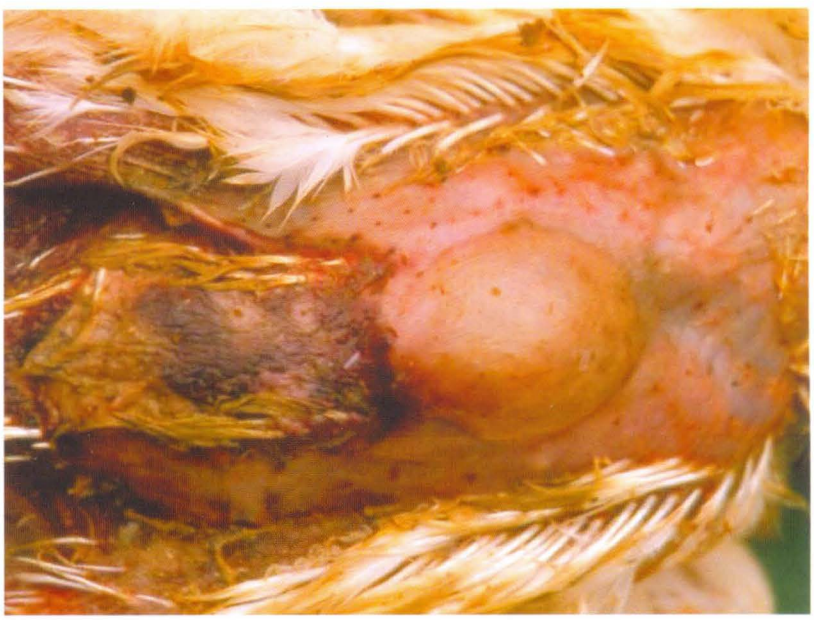
151, 152, 153. Most outbreaks are encountered in broilers at the age of 4 - 8 weeks. The disease is also observed in stock layer hens at the age of 6 - 20 weeks and broiler parents at the age of 20 weeks. The outbreaks are frequently observed in extremely wet and warm premises. The gangrenous dermatitis affects birds while still alive. In more s evere cases, the gangrene could begin and involve the skin of the head, neck and the breast. Affected skin is macerated or totally necrotic, resulting in exposure of underlying tissues in a number of cases.



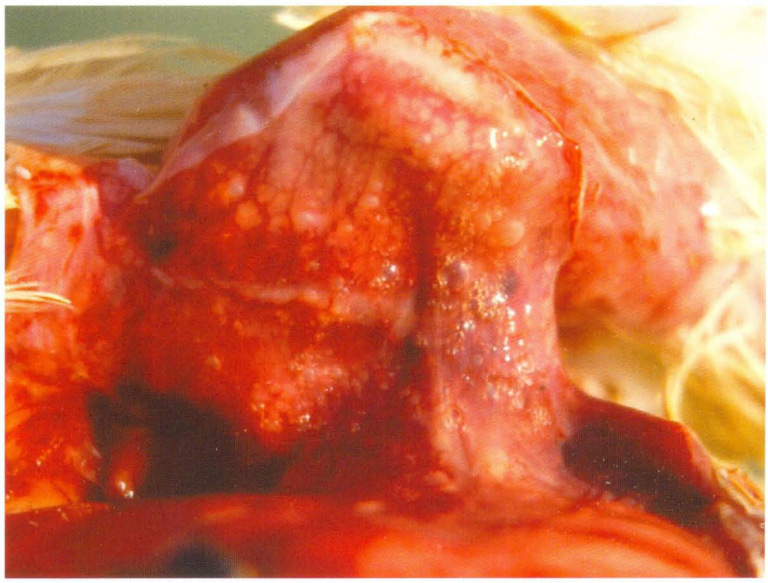




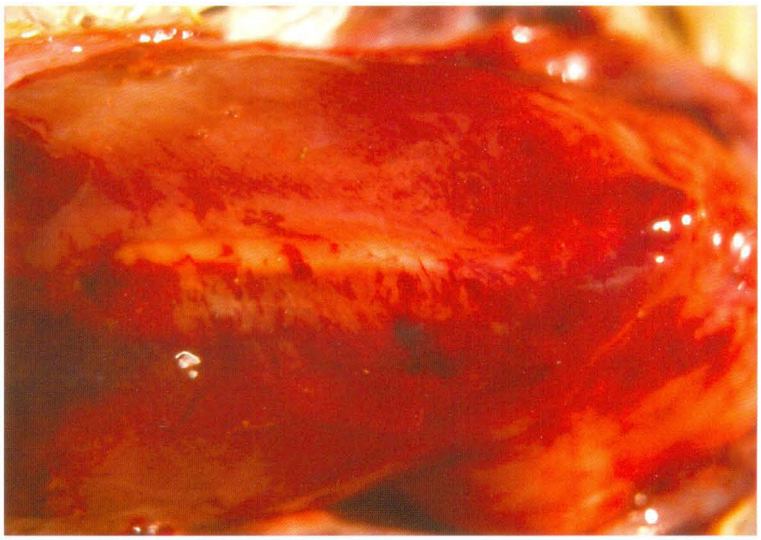
154. The GD agents are *Cl. septicum*, *Cl. perfringens* type A and *Staphylococcus aureus*, independently or in combination. The associated infection is more severe. The skin lesions are often crepitating and are detected in the regions of breast, abdomen, back or wings in both alive and dead birds.

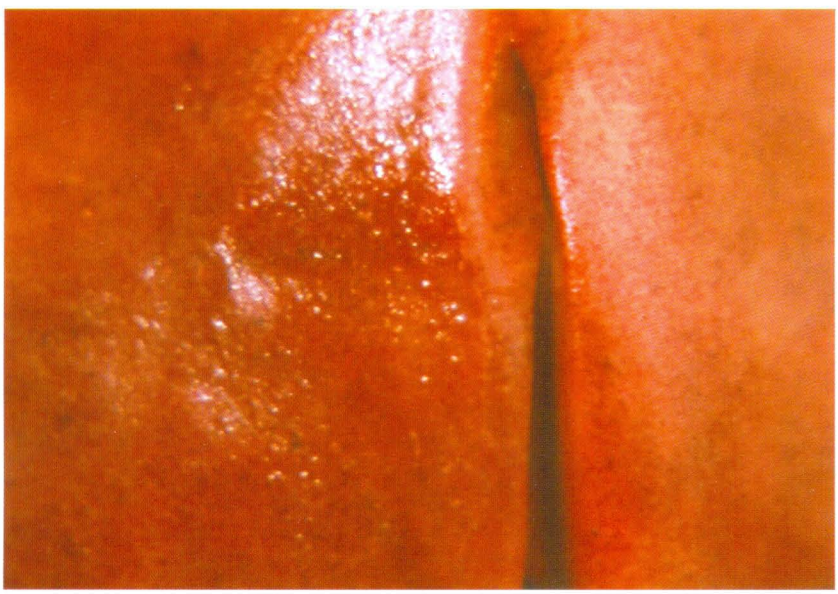


155. Under the affected skin, extensive haemorrhagic oedemas with or without gas (emphysema) are discovered. The clostridia are encountered in soil, faeces and the intestinal content. Staphylococci usually inhabit the skin and mucous coats of birds.

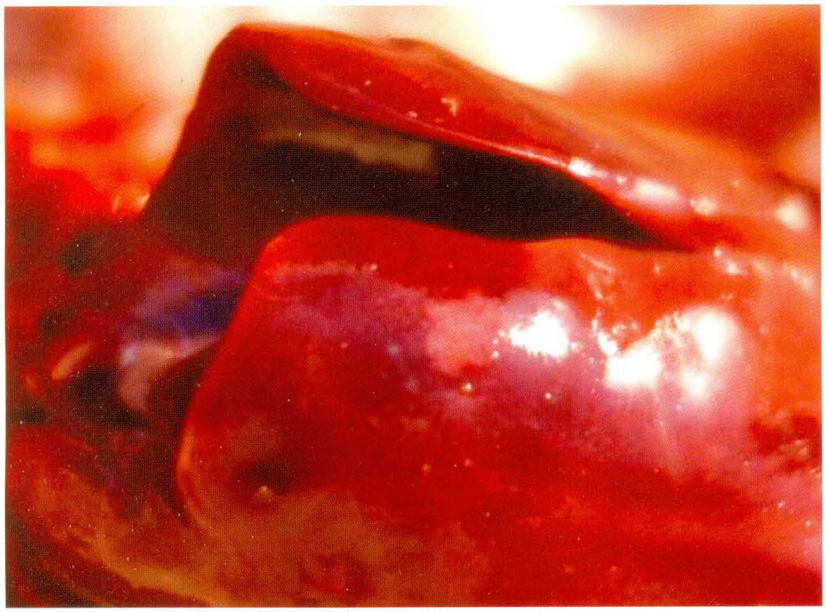


156. Underlying muscles are haemorrhagically infiltrated and also could contain gas among the muscle fibres. The increased susceptibility of birds to GD is related to immunodeficiency states secondary to CIA, IBD, IBH etc. Other predisposing factors are aflatoxicoses, the unbalanced or deficient diet or poor hygiene. The skin wounds are the entrance door of the infection.





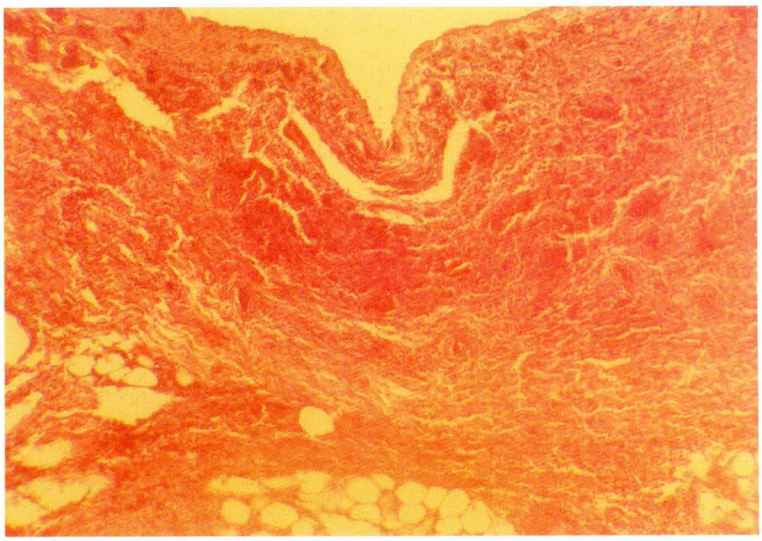
157. In most cases, no changes in viscera are observed. Rarely, gas bubbles could be found out in the liver. **The diagnosis** is confirmed by imprint preparations or histological cross sections. The bacterial agent could be isolated from cellulitic zones.



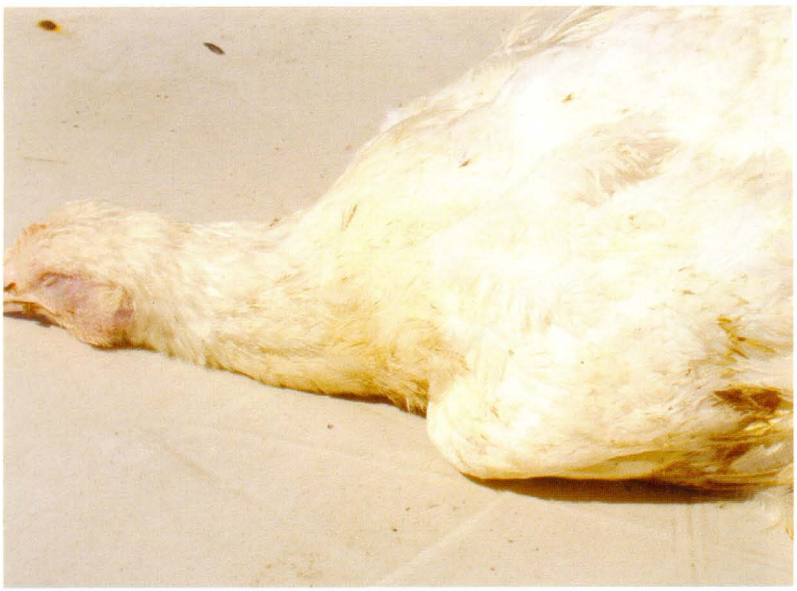
158. Single or multiple necroses are rarely seen in the liver.

Prevention - the factors leading to skin injuries (cannibalism, mechanical traumas etc.) should be minimized. Successful approaches are the immunoprophylaxis programmes in breeder flocks aiming at prevention and reduction of possible immuno-suppressive states.

159. Microscopic lesions are characterized by oedema, emphysema, hyperaemia, haemorrhages and necroses in the subcutaneous tissues. GD could be effectively treated by tetracyclines: doxyxycline hydrochloride, erythromycin thiocyanate, chlortetracycline and copper sulfate in the water.



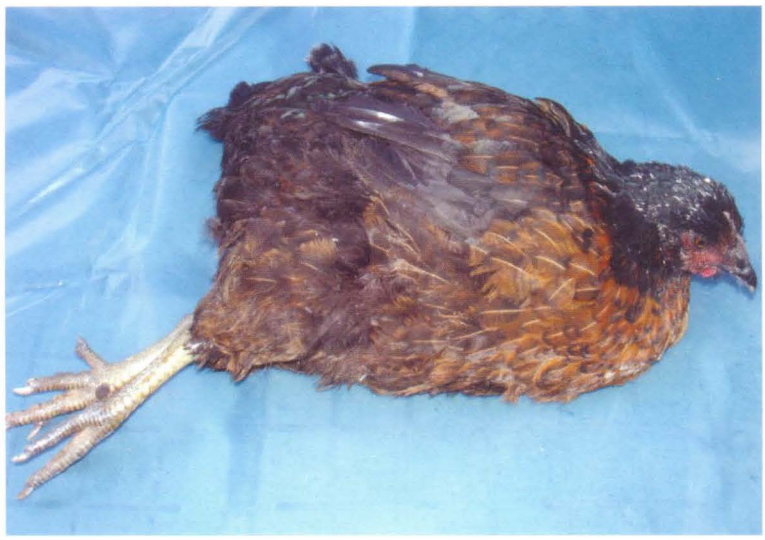
BOTULISM



160. Botulism is an intoxication caused by the toxins of *Clostridium botulinum*. With the exceptions of vultures, most birds are susceptible. The clinical signs appear within a few hours to several days. Flaccid paresis of legs, wings, necks and eyelids is observed. The paresis is rapidly progressing to paralysis and the birds fall into a deep coma with neck and head typically extended forward. There are no typical gross lesions. The treatment with selenium, vitamins A, D and E as well as with some antibiotics as chlortetracycline, bacitracin etc. could reduce mortality rate.

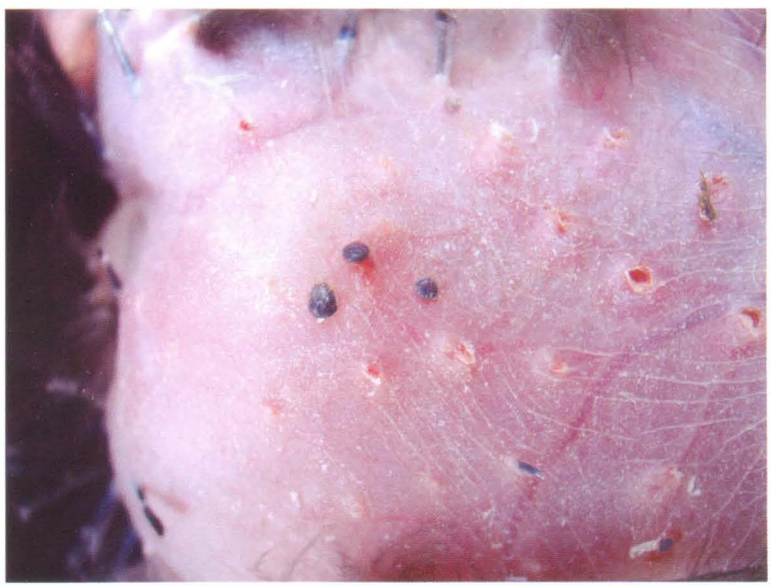
SPIROCHAETOSIS

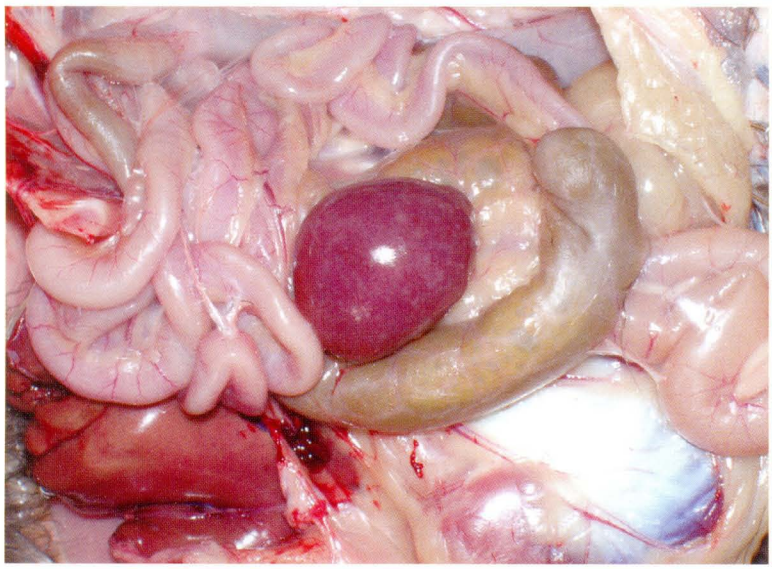
161. Spirochaetosis is a septicaemic disease characterized by depression, progressive paresis, paralysis and inflammatory necrobiotic changes in parenchymal organs and the gastrointestinal tract. The birds are depressed, cyanotic; greenish diarrhoea with considerable amounts of urates is observed.



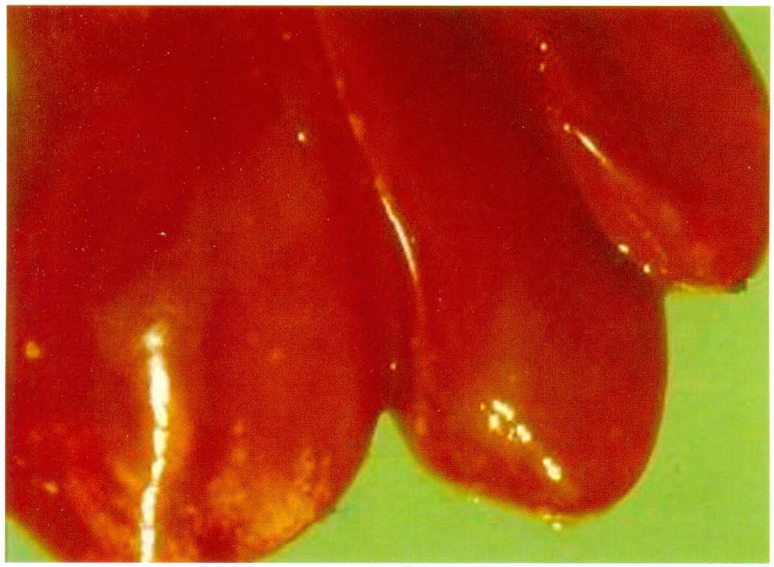
162. The transmission of spirochaetosis is related to the distribution of the fowl tick *Argas persicus* that is the reservoir and main vector. Hens, turkeys, geese, ducks, pheasants etc. from all ages are susceptible. At a later stage, paresis and paralysis are developing, the birds become somnolent and comatose.

163. After removal of plumage, the ticks, adhered to the skin, are discovered. The aetiological agent is *Borrelia anserina*, a highly motile spirochaete with 5-8 coils. The organism is not resistant outside the host and could exist only in some vector.





164. A typical finding for spirochaetosis is the enlarged spleen with marble-like appearance. *A. persicus* remains infective for over 430 days and transmit spirochaetes to its progeny. Spirochaetes could be identified in Giemsa-stained blood smears. In the late stage of the disease, spirochaetes could not be detected.

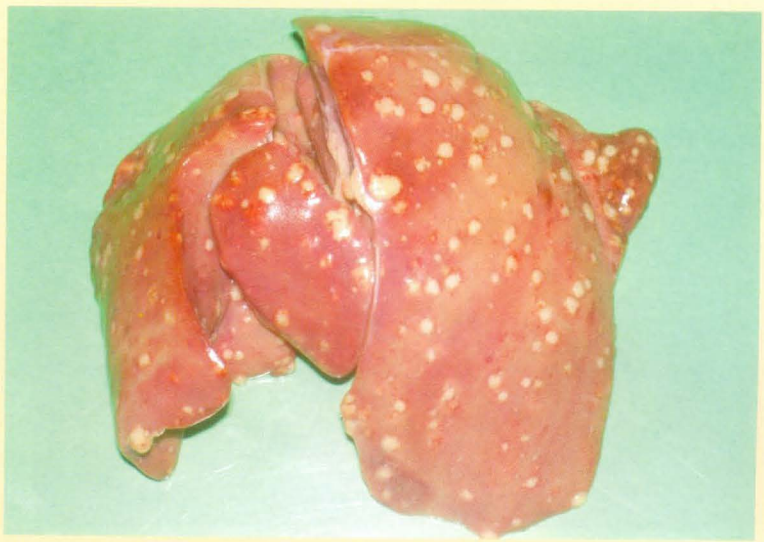


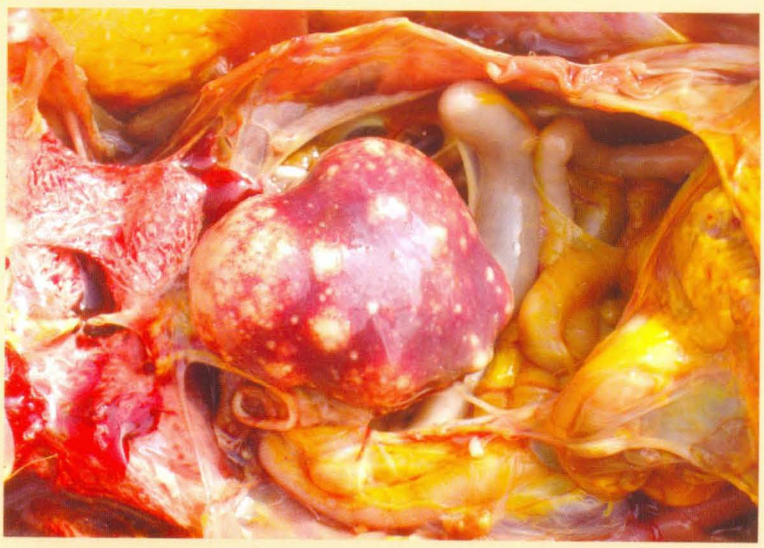
165. The liver is often enlarged, mottled with necroses of a various size and marginal infarctions. Usually, bile-coloured mucoid enteritis is observed. The arsenics as well as some antibiotics including tylosin, tetracycline and penicillin are effective for treatment of infected birds. The application of vaccines is successful, but the acquired immunity is short and revaccinations are needed.

AVIAN TUBERCULOSIS

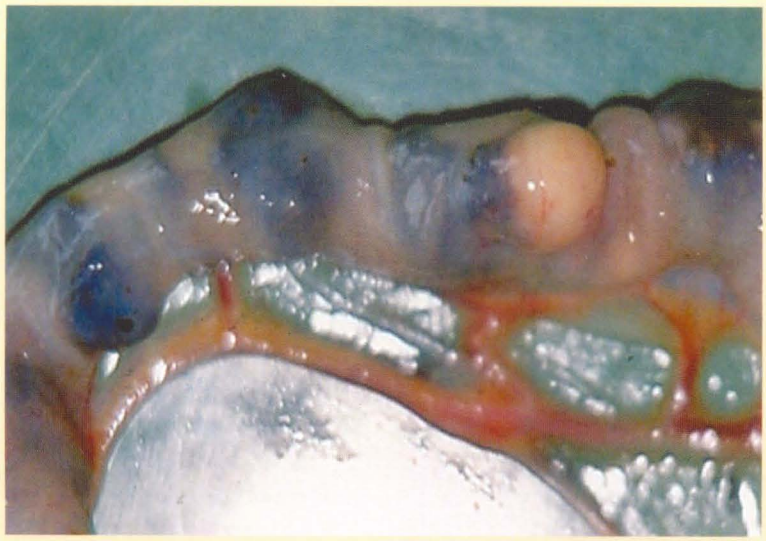
166, 167, 168. Avian tuberculosis is a chronic infectious disease characterized by the formation of granulomatous lesions in viscera, a progressive weight loss and death. It is usually encountered sporadically in birds reared in small yards, zoos and is a problem among caged exotic birds.

A progressive weight loss to cachexia, diarrhoea and pallor of the skin of the comb, wattles and the face are observed. The parenchymal organs (especially the liver and the spleen) are enlarged and within, granulomas (tubercles) of a various size are found. Pulmonary lesions are rarely encountered.



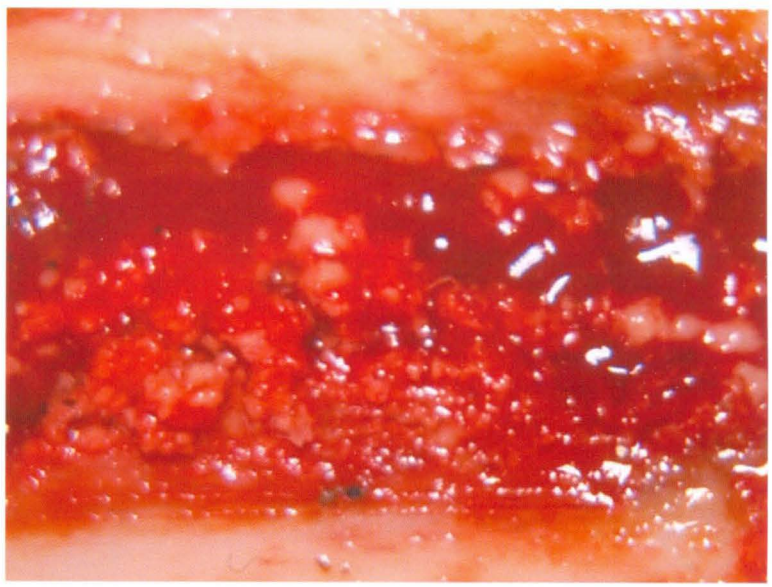


169, 170. The aetiological agent is *Mycobacterium avium*, a very resilient and acid-resistant micro-organism. It is resistant to temperature changes, drying, pH changes, to many disinfectants and survives in the soil for years. Along the small intestine, single or multiple subserously prominating tubercles are detected.

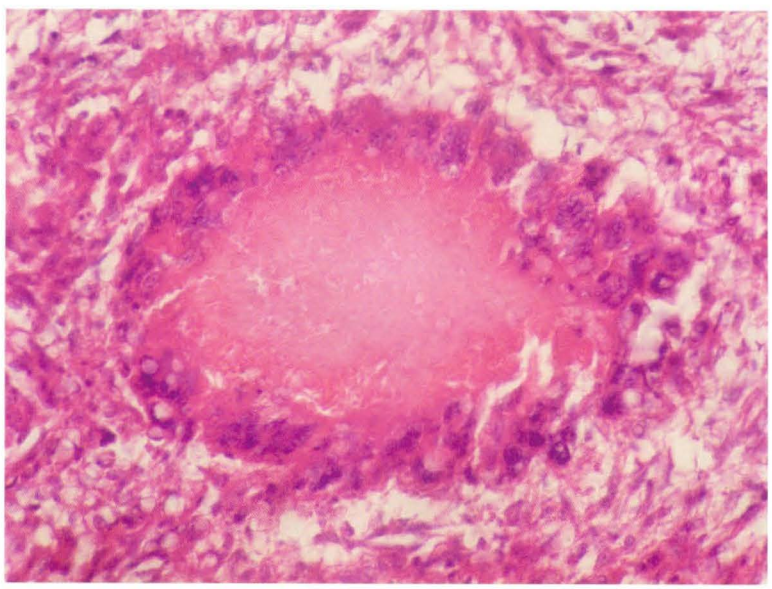


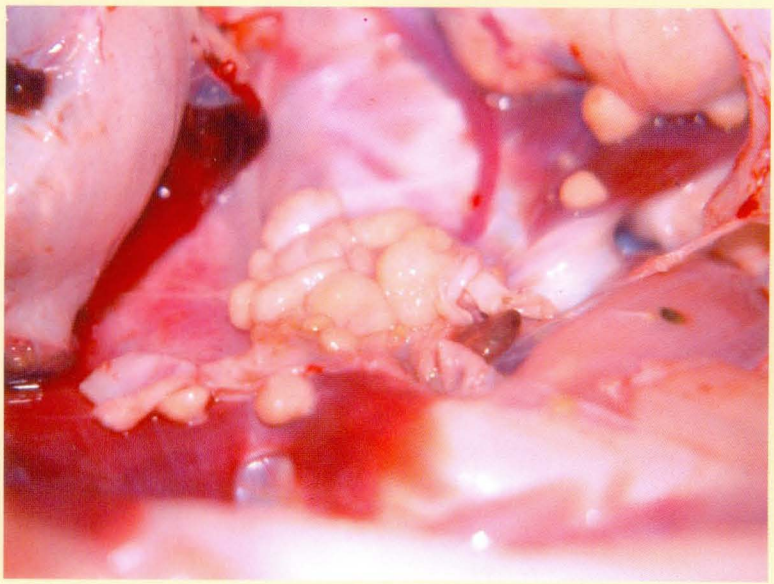
After necrotization of some tubercles, pseudodiverticula that are constantly shedding viable mycobacteria are formed. The transmission of the agent is realized mainly via intake of contaminated forage, water, litter and soil.

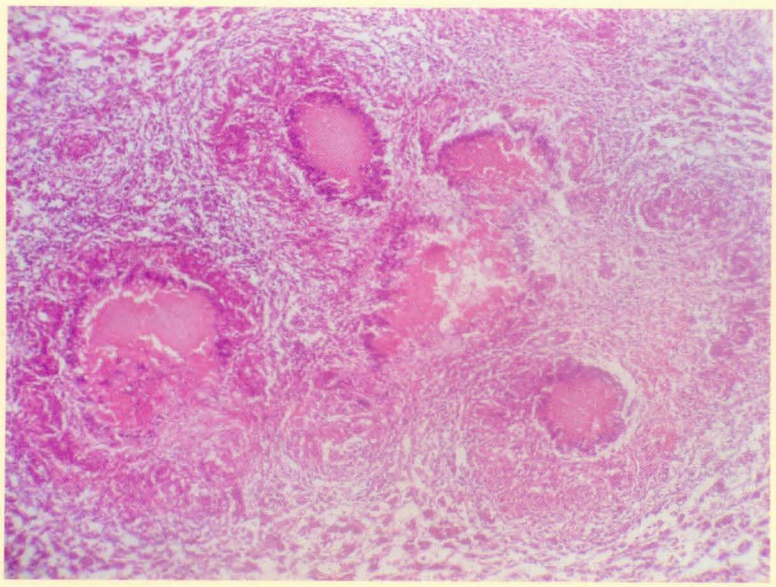
171. In advanced cases, tubercles could be observed along the bone marrow of the femur or the tibia.



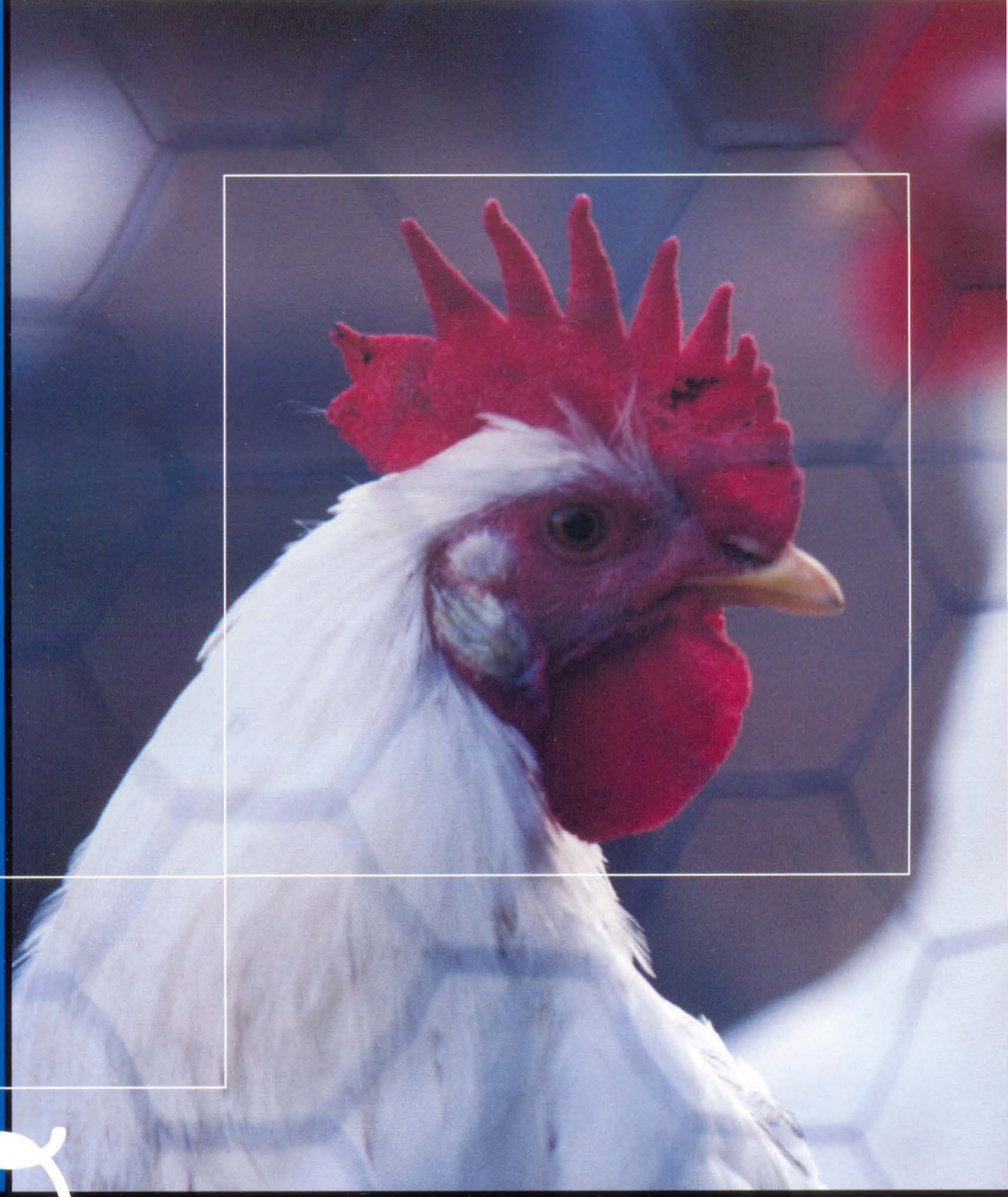
172. The main mass of the tubercle is composed of epitheloid cells, and peripherally, there are foreign body-type multinucleated giant cells. In older granulomas, central coagulation or caseous necroses are present.





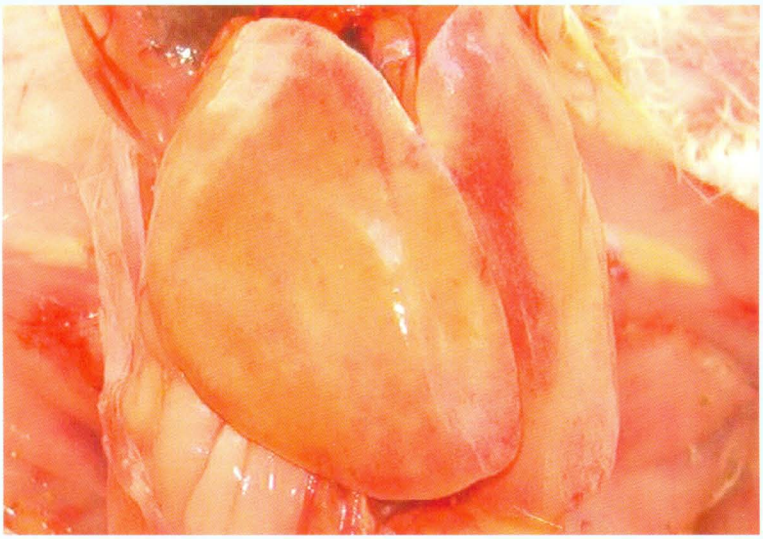


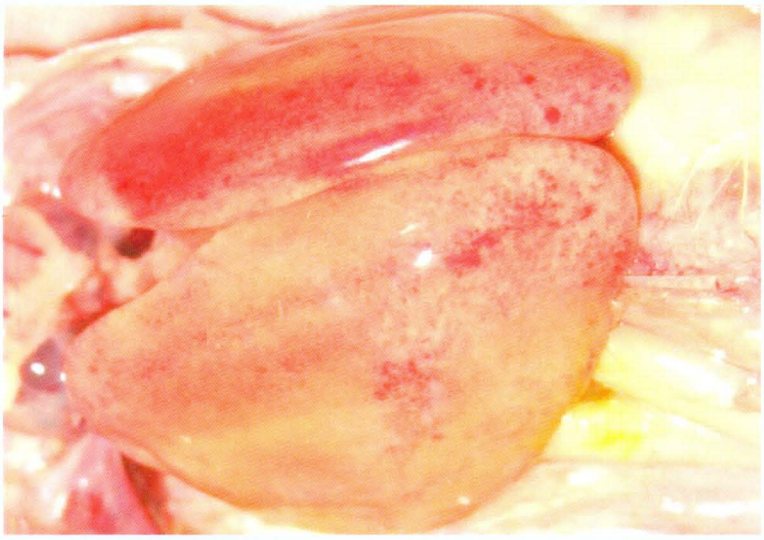
173, 174. Characteristic features of avian tuberculosis are conglomerate tubercles (gross and histological appearance). **The diagnosis** is based upon the complex evaluation of history, persisting lethality in adult flocks and the pathoanatomical findings. Avian tuberculosis should be differentiated from neoplastic diseases, coligranuloma (Hjarre's disease), pullorum disease etc. The treatment is not advised, as the disease is contagious for men.



Viral diseases

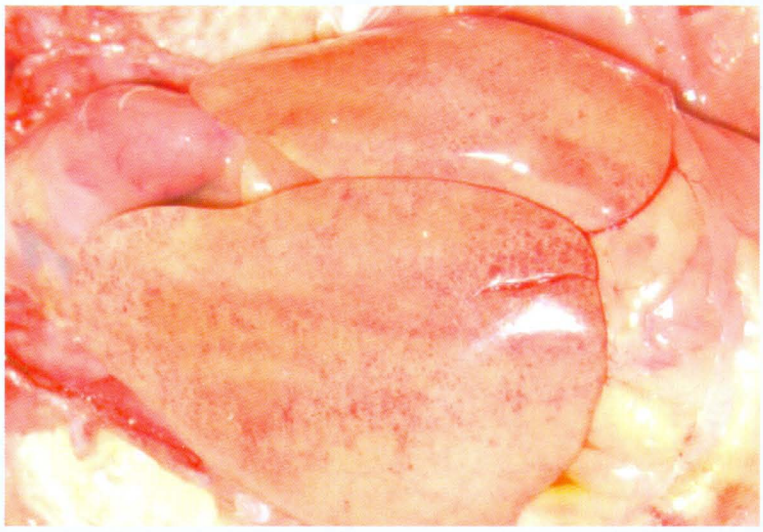
VIRAL INCLUSION BODY HEPATITIS

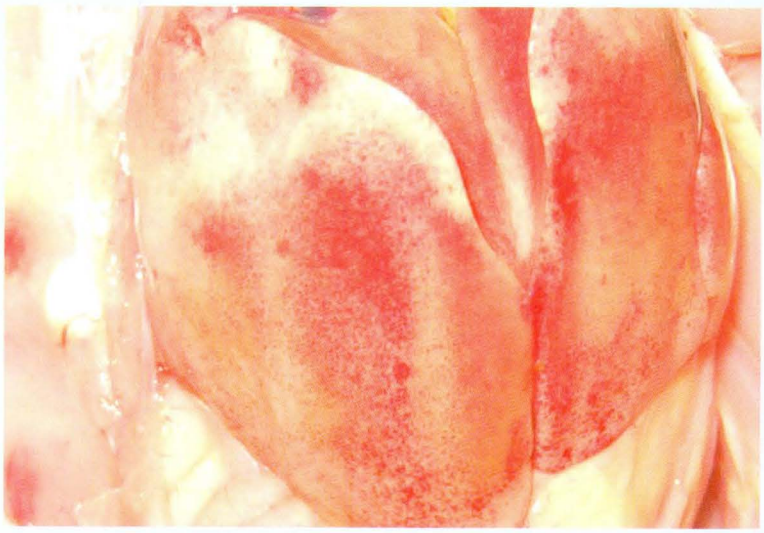




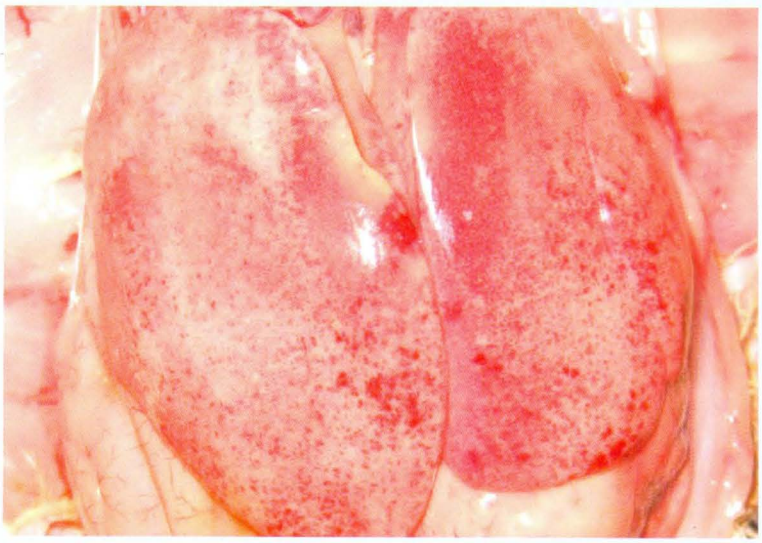
175, 176. The viral inclusion body hepatitis (IBH) is an adenovirus infection characterized by haemorrhages and dystrophic necrobiotic changes in the liver and kidneys, accompanied by intranuclear inclusion bodies. A characteristic macroscopic lesion is the enlarged, dystrophic liver with yellowish colour and crumbly texture.

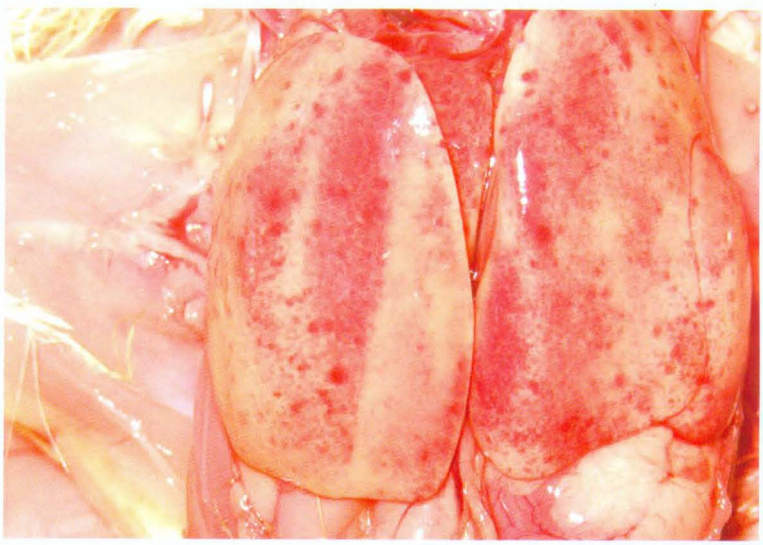
177, 178. IBH outbreaks are encountered primarily in meat type chickens, most commonly at the age of 3-8 weeks. IBH often occurs as a secondary infection to immunodeficiency resulting from other diseases (IBD, CIA). On the background of dystrophic liver changes, haemorrhages of various intensity and size are outlined, thus creating a variety of liver lesions.

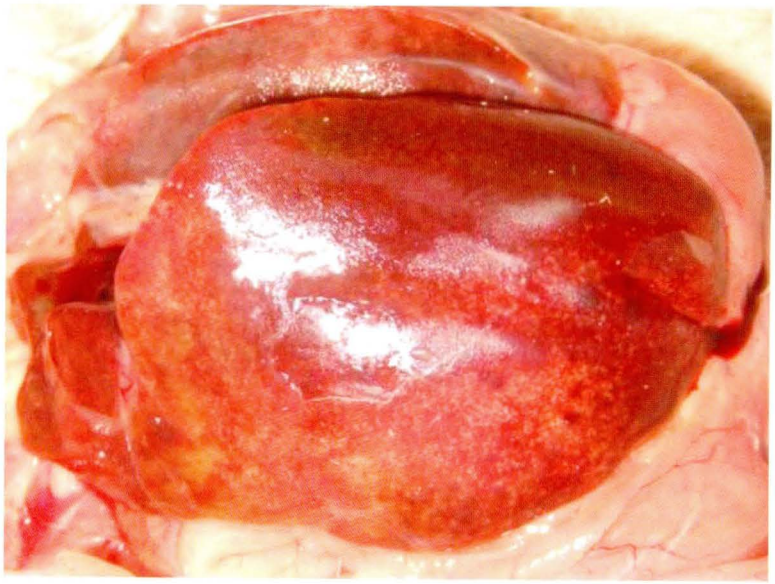




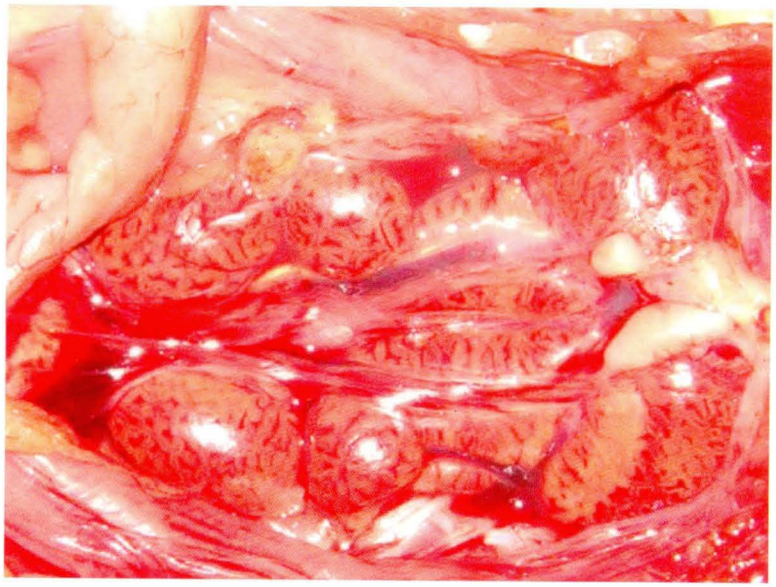
179, 180.
Throughout IBH outbreaks, several serotypes from the 12 known avian adenoviruses (AAVs) of group I are isolated. The sick chickens carry the virus in their excreta, kidneys, tracheal and nasal mucosa. The virus is resistant to many environmental factors and could be easily transmitted by a mechanical route. The transmission of adenoviruses is realized vertically by breeder eggs and horizontally, via excreta (mainly faeces). In a number of cases, the dominating lesion is the massive mottled or striated haemorrhages of the liver.



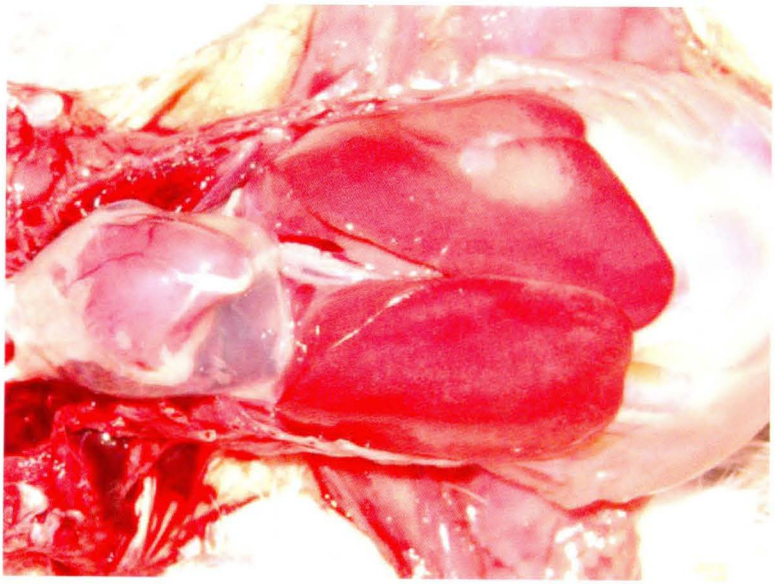




181. IBH is characterized by a sudden onset and a sharply increased death rate that reaches peak values by the 3rd - 4th day and returns back within the normal range by the 6th - 7th day. The total death rate is usually under 10% but sometimes could attain 30%. More rarely, macroscopically visible necrotic foci could be detected in the liver.

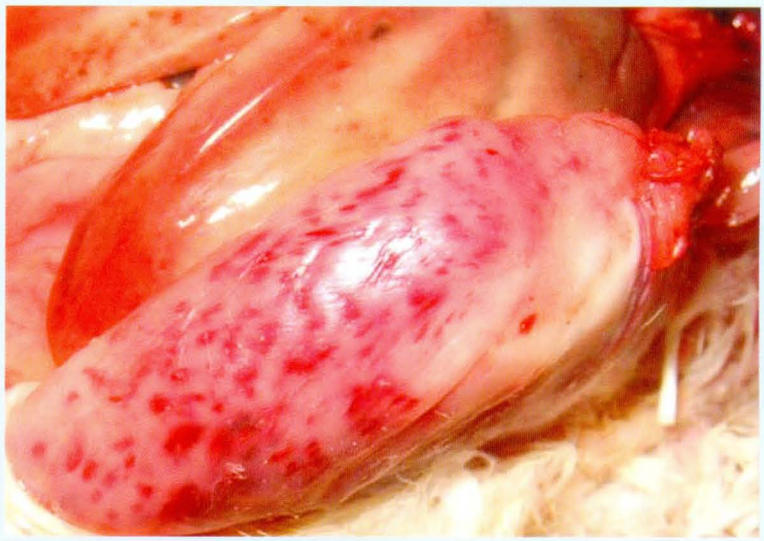


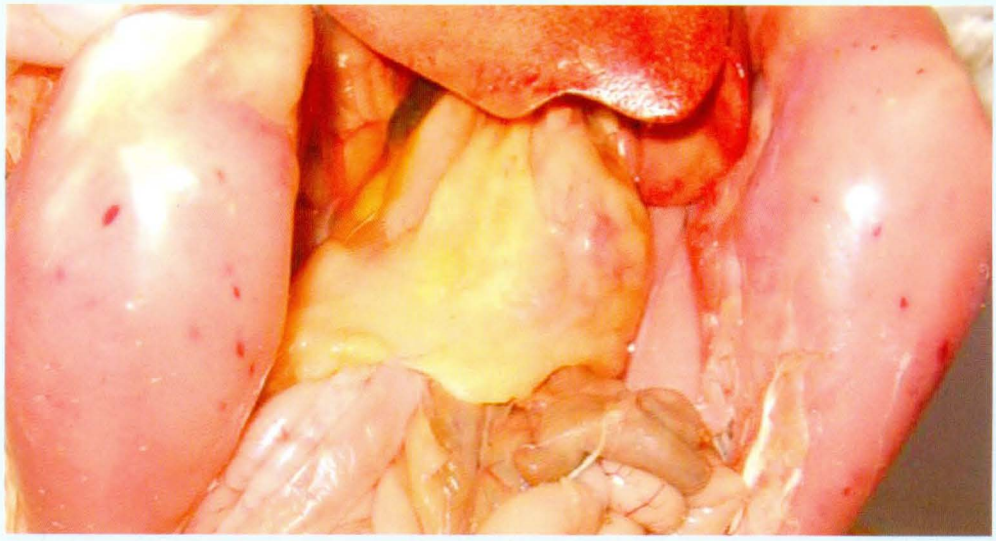
182. The kidneys are enlarged, pale and mottled with multiple haemorrhages.

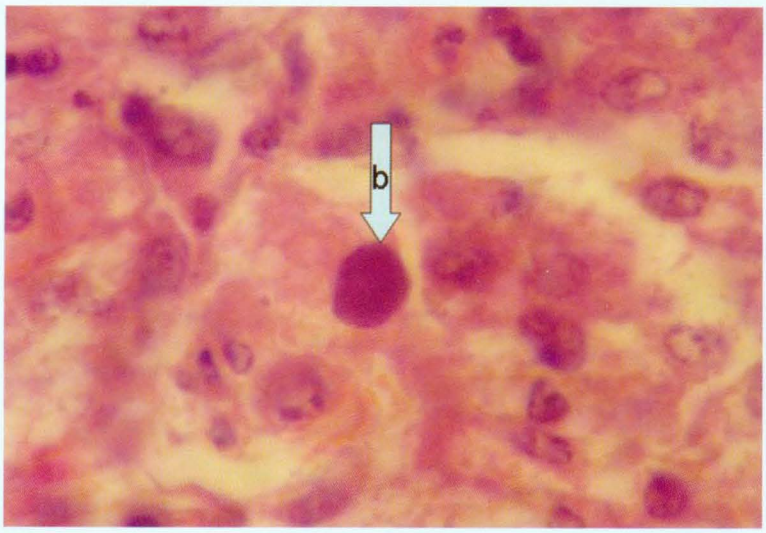


183. In many instances of IBH, the amount of pericardial fluid is increased (hydropericardium).

184, 185. Clinical signs could be observed only several hours prior to death occurrence. They consist in pale comb and wattles, depression and apathy. Sometimes, the skin is icteric. Often, ecchymoses and striated haemorrhages in skeletal muscles are observed.

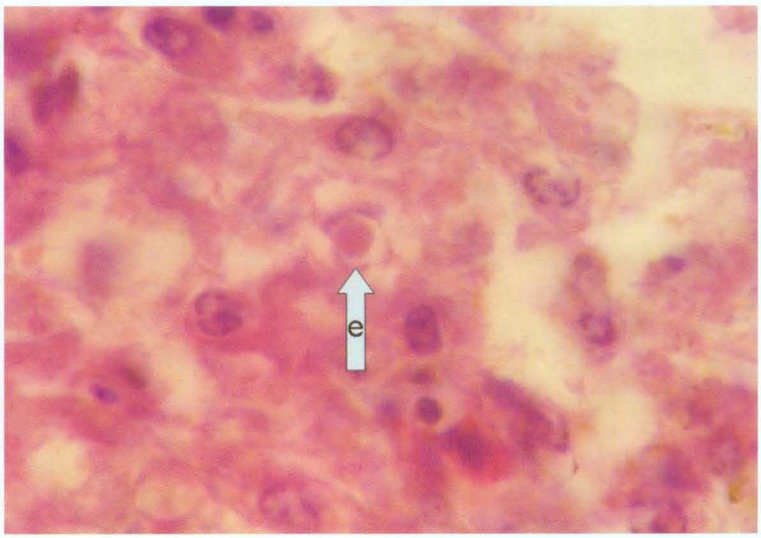




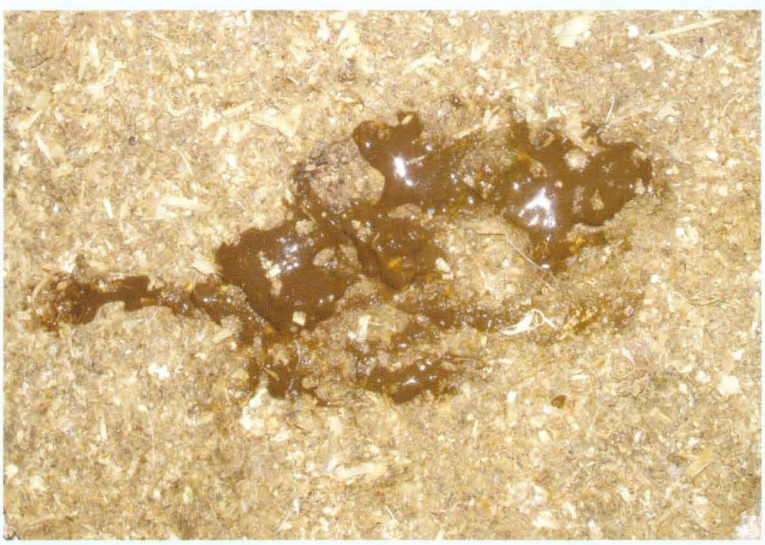


186[A], 186[B]. Microscopically, extensive dystrophic changes and necroses of liver parenchyma are detected. In the nuclei of hepatocytes, basophilic or eosinophilic inclusion bodies are detected. The basophilic inclusion bodies are usually dense and occupy the entire nuclear inner space **(arrow b)**, whereas the eosinophilic ones are round or irregularly shaped and surrounded by a light halo **(arrow e)**. The diagnosis is based upon the typical gross lesions and the history records.

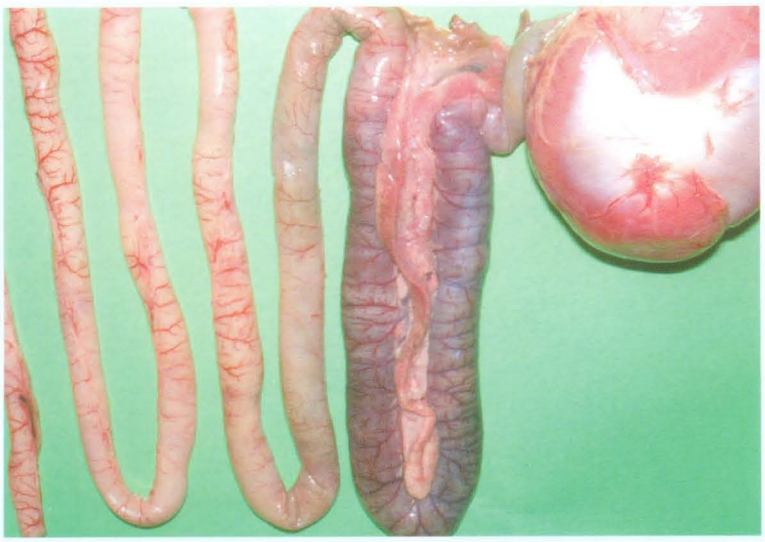
A principal approach in IBH diagnostics is the histological investigation that helps to detect the intranuclear inclusion bodies. IBH should be distinguished mainly from IBD and chicken infectious anaemia (CIA). With regard to IBH prevention and control, the eggs of broiler parent flocks, where the disease is consecutively appearing in the progeny, should not be used for hatching. The access of wild birds should be prevented as they are potential carriers and distributors of the virus. The most important steps in IBH prevention are the control of IBD and CIA. There are neither vaccines, nor an effective treatment.

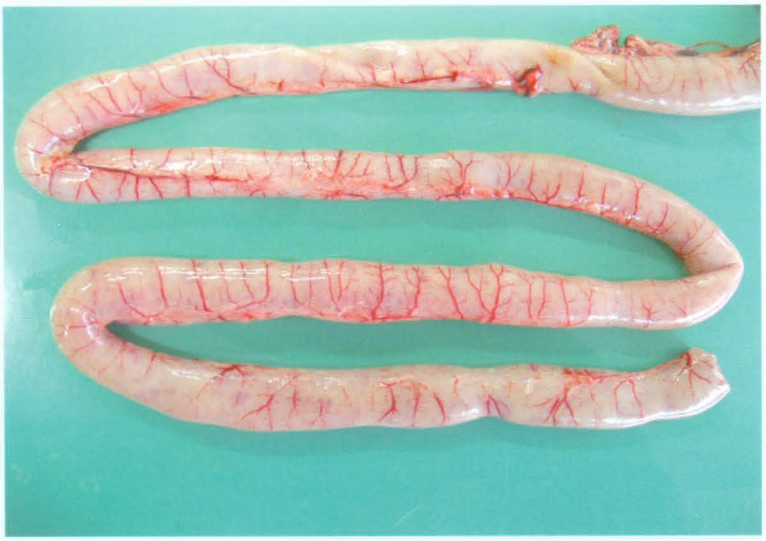


HAEMORRHAGIC ENTERITIS OF TURKEYS



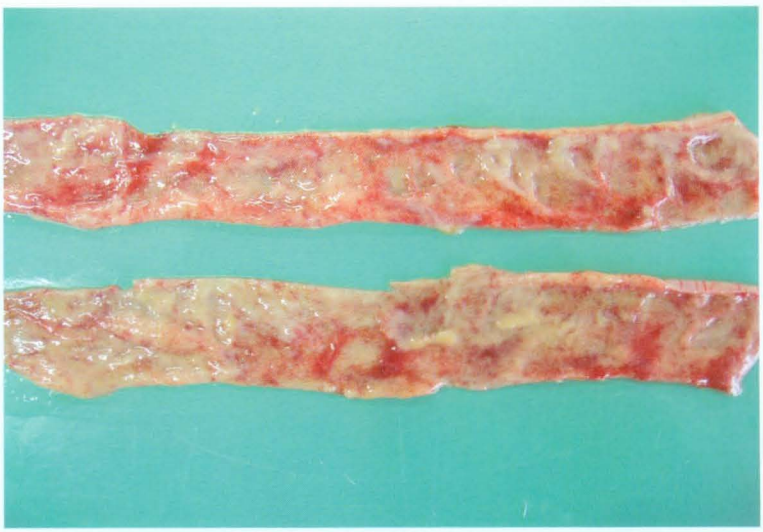
187, 188. The haemorrhagic enteritis (HE) is an acute viral disease in young turkeys, characterized by a sudden onset, bloody faeces and various, but often high death rate. Blood discharge from the vent (187), fresh blood in faeces or melena (188) could be observed.



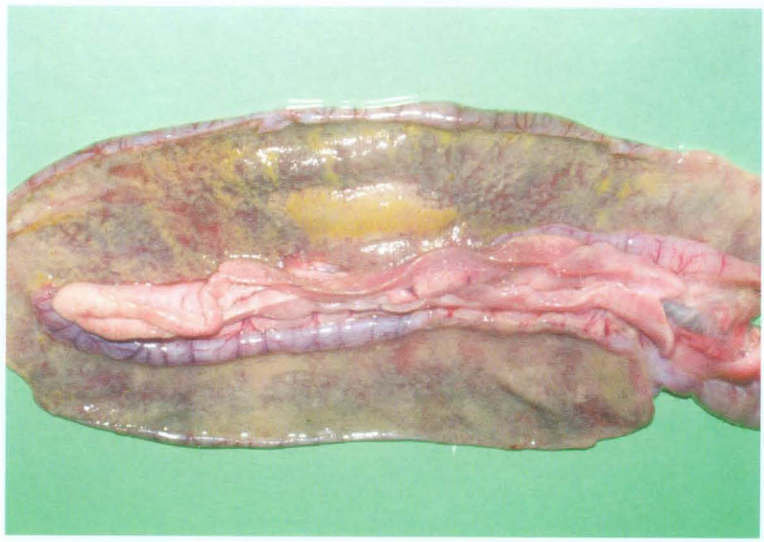


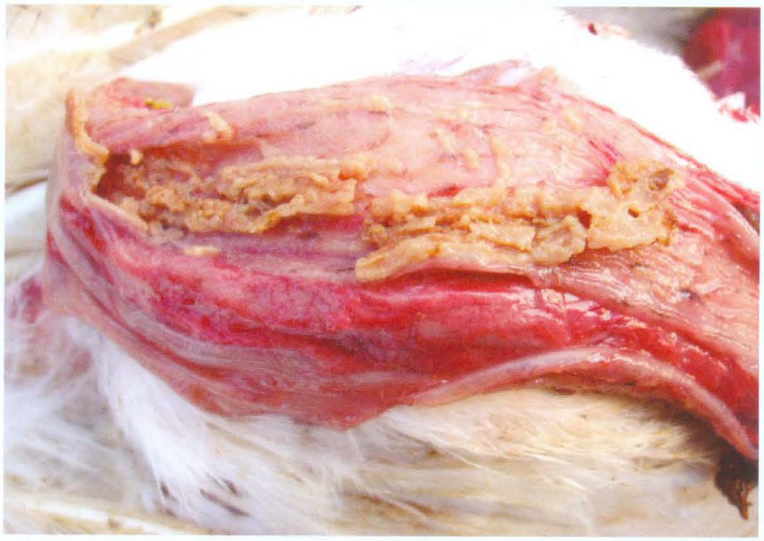
189, 190. HE is observed in turkeys at the age of 6 - 11 weeks but most commonly at the age of 7 - 9 weeks. As an exception, HE could be observed in turkeys under 4 weeks of age, presumably due to maternal antibodies. The small intestine, especially the duodenum, has a dark red colour and ramiform blood vessels prominating under the serous coat, and sometimes, haemorrhages are seen through the intestinal wall.

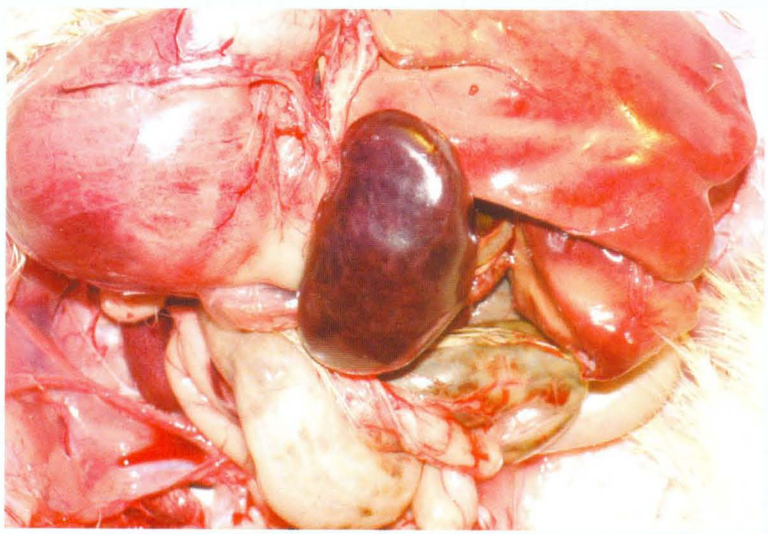
191, 192, 193.
HE is characterized by rapidly progressing clinical signs and disease course of about 10 days. The peak death rate is reached by the middle of the period. The entire death rate varies between 2-3% and 5-10% but could reach 60% as well.



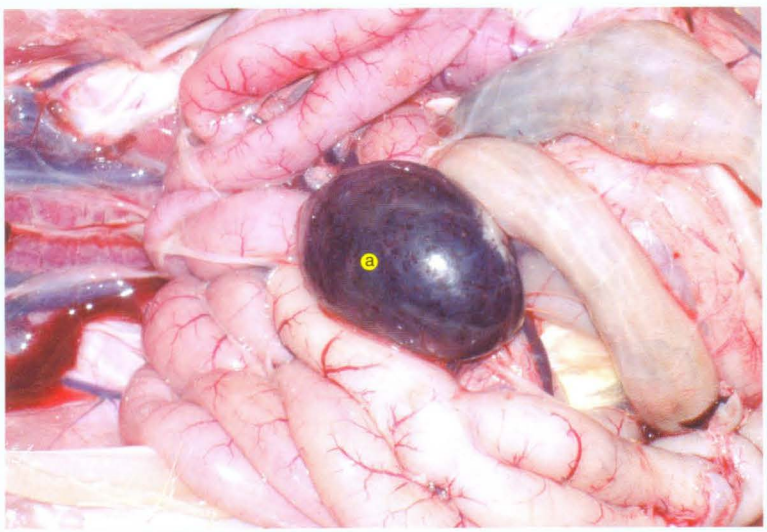
The turkeys are depressed, a sharp reduction in forage and water consumption is seen. The extent of lesions of the interstitial mucosa varies from hyperaemic to haemorrhagic (191), severe haemorrhagic or fibrinous necrotic inflammation (192 & 193).



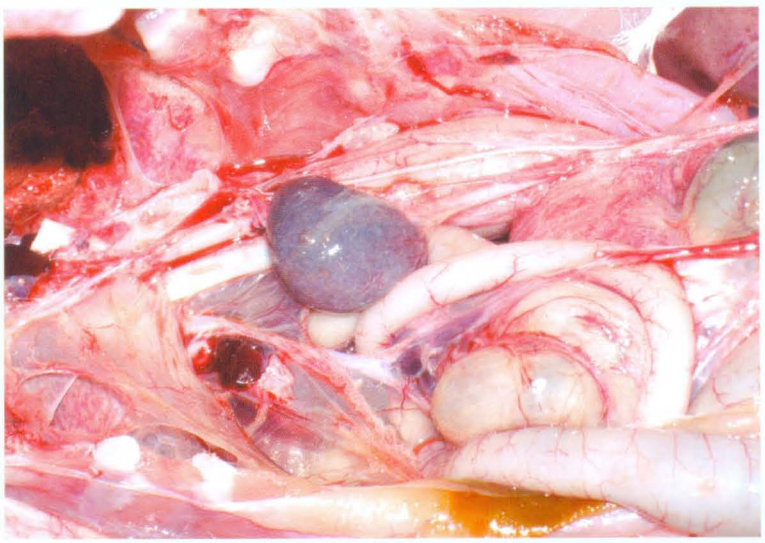


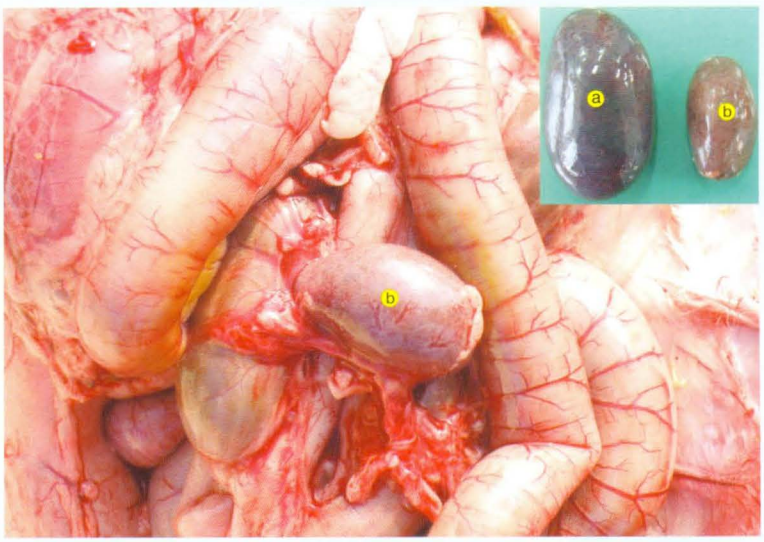


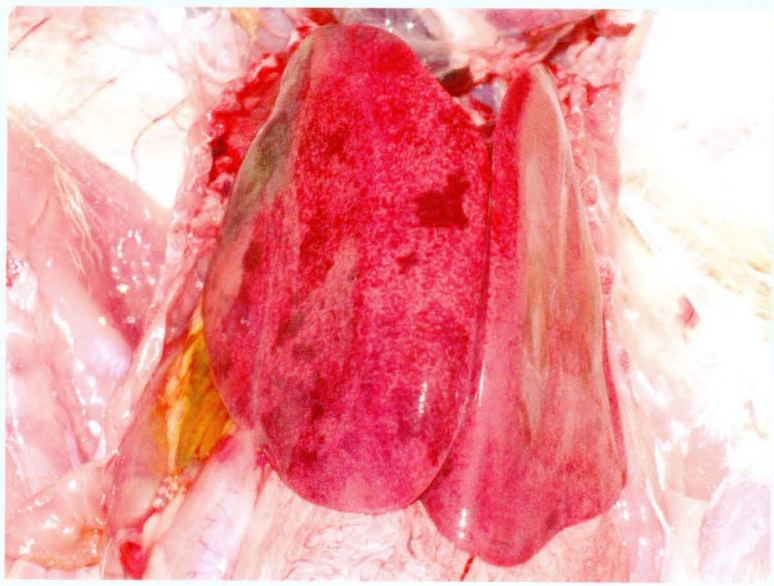
194, 195. The spleen of infected birds is typically enlarged, haemorrhagic, crumbly, mottled or marble-like. The aetiological agent of HE is an adenovirus of group (type) II (HEV). The viruses of marble spleen disease in pheasants (MSDV) and avian adenovirus splenomegaly (AAS) that are serologically similar, belong to the same group.

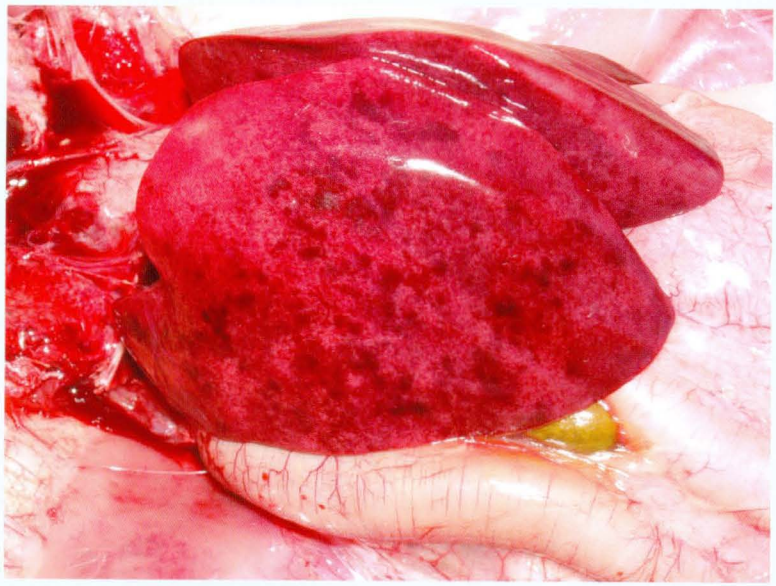


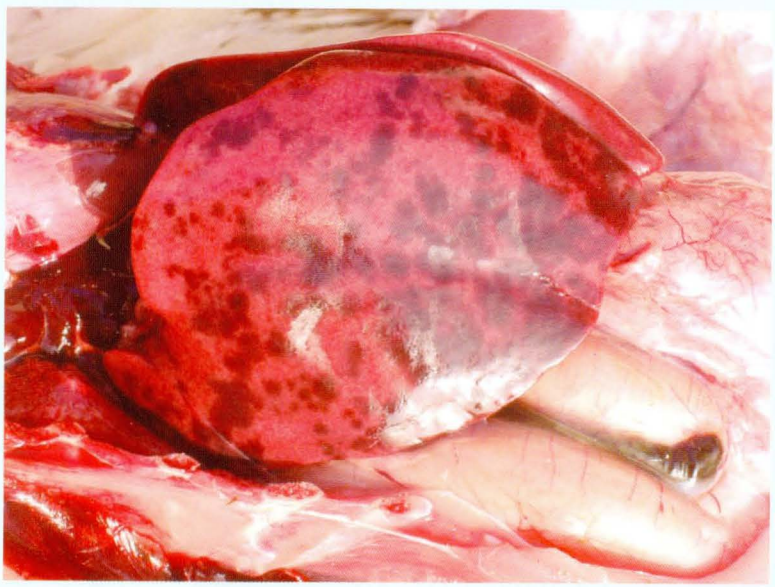
196, 197. Later, the spleen reduces its size 2-3 times and acquires a specific silvery-grey colour. The serological studies show that HEV is widely distributed among adult turkeys. There is no evidence for vertical transmission. HEV is spread horizontally by ingestion of faeces of infected turkeys. Contaminated litter, equipment, shoes, other birds, rodents etc. are also important in the transmission of the infection.





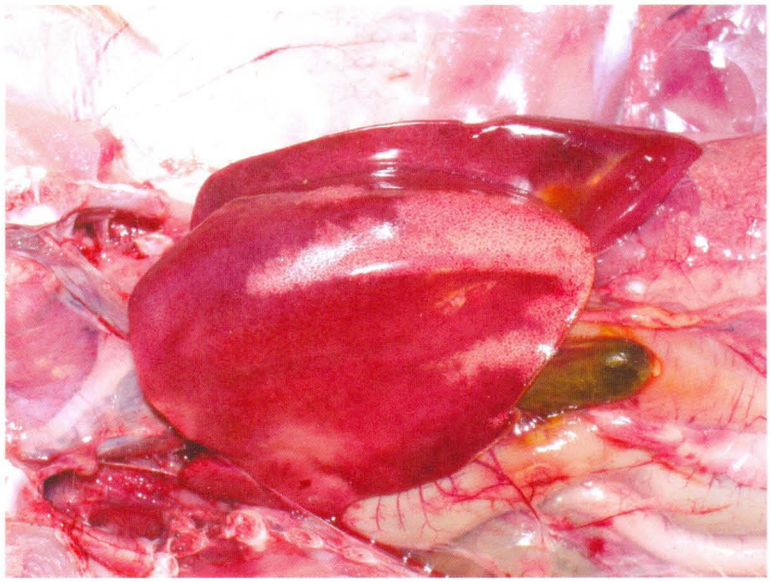






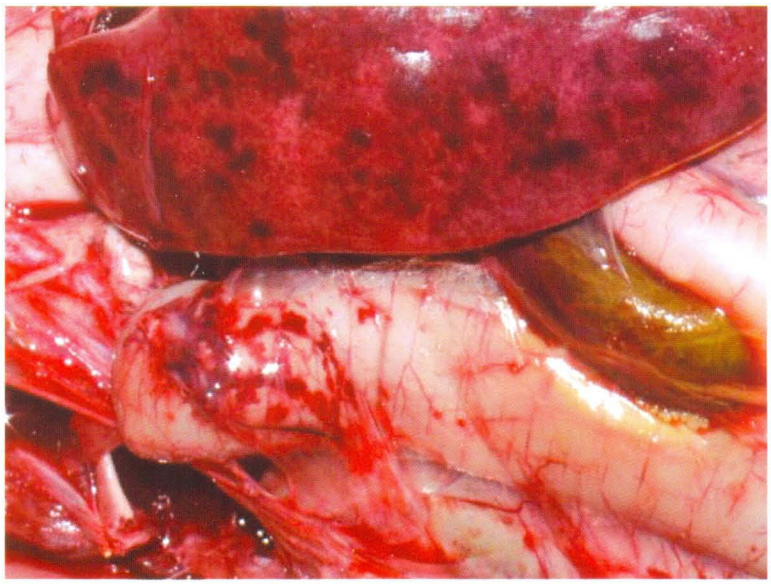
198, 199, 200.
The liver is enlarged, crumbly and mottled with multiple haemorrhages, varying from petechiae to ecchymoses. Dead birds are in a good condition, with pale skin. Once having appeared in the farm, the infection is often recurring in other flocks. The HEV infection in turkeys results in a transient immunosuppression and consequently, a secondary *E. coli* septicaemia.

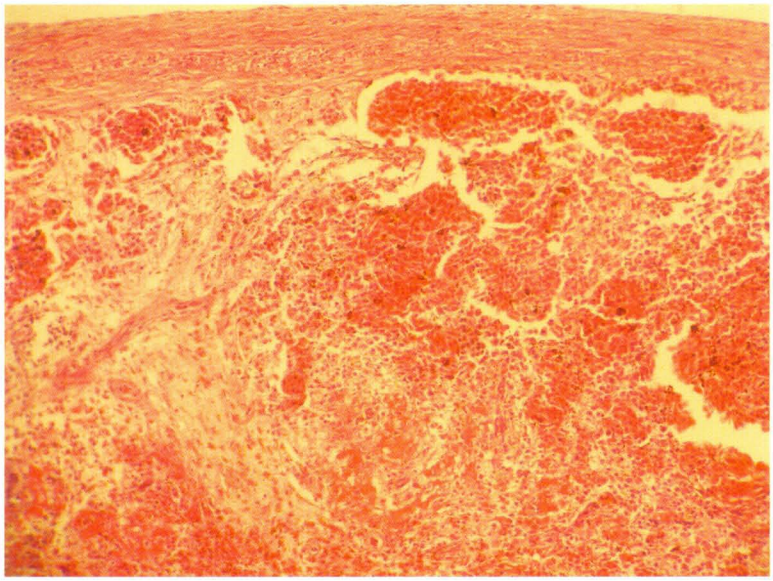
201. Less frequently, extensive necrotic foci, sometimes haemorrhagically infarcted, could be detected in the liver.



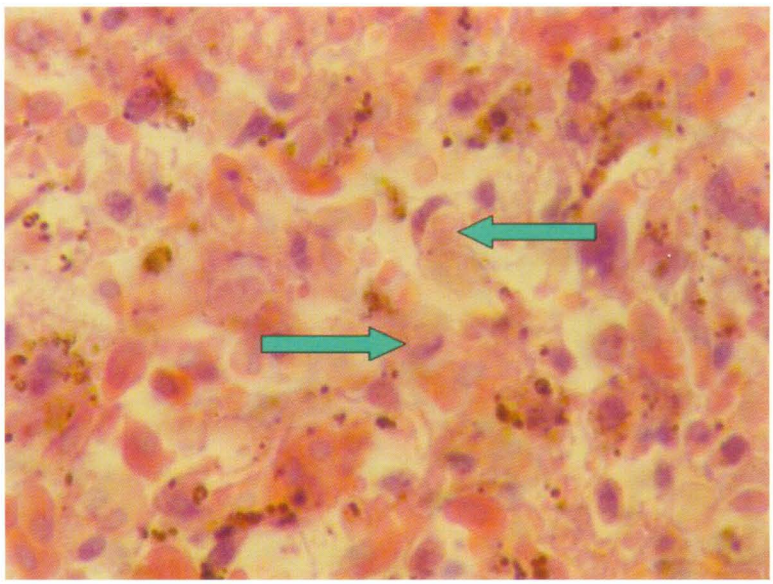
202. The kidneys are enlarged and haemorrhagic.

203. In many instances, the small intestinal and gastric serous coats are encompassed by petechial or striated haemorrhages.

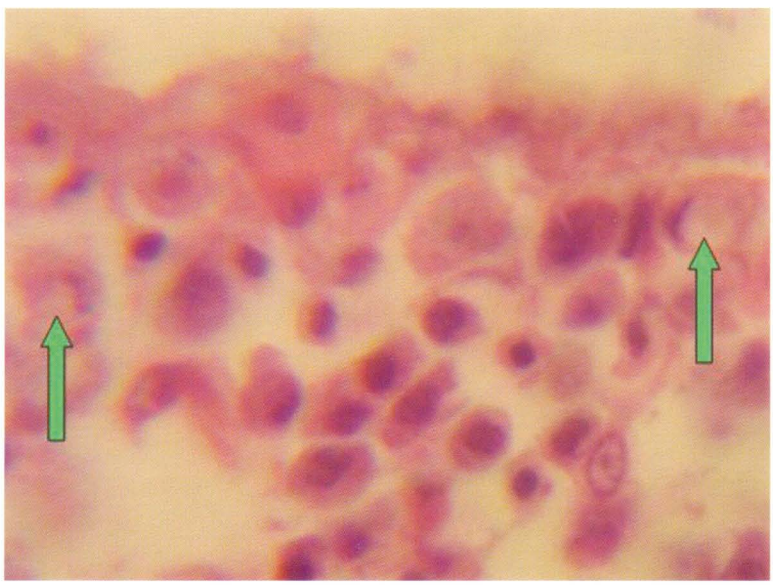




204. Microscopically, hyperplasia of the white spleen pulp, necroses, subcapsular and periarteriolar haemorrhages are observed.

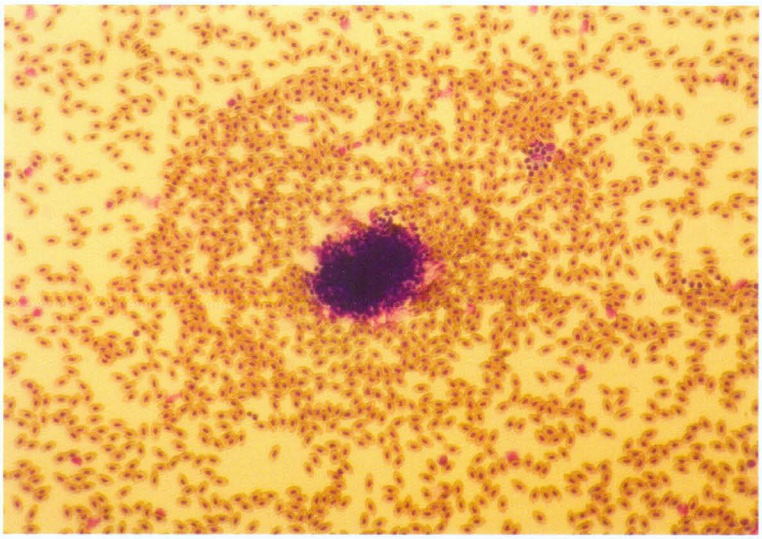


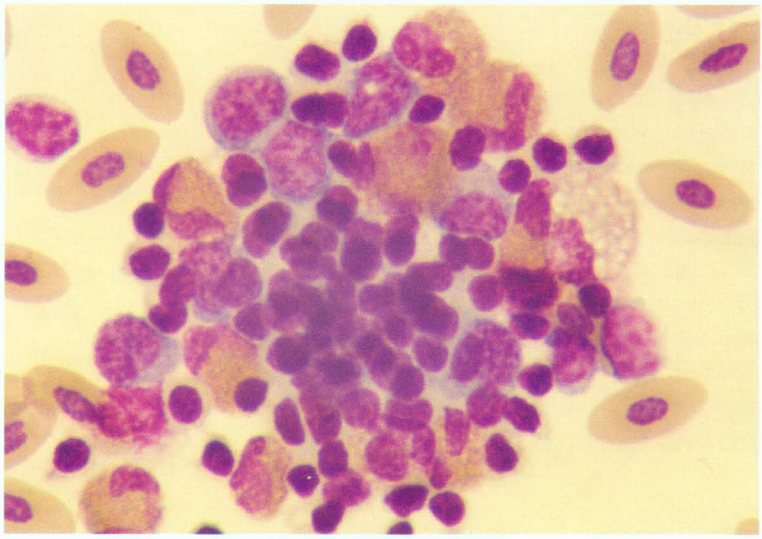
205. A characteristic diagnostic sign is the discovery of large acidophilic, rarely basophilic intranuclear inclusion bodies in the reticuloendothelial cells. The displaced and condensed nuclear chromatin pattern around the inclusion bodies often looks like a crescent.



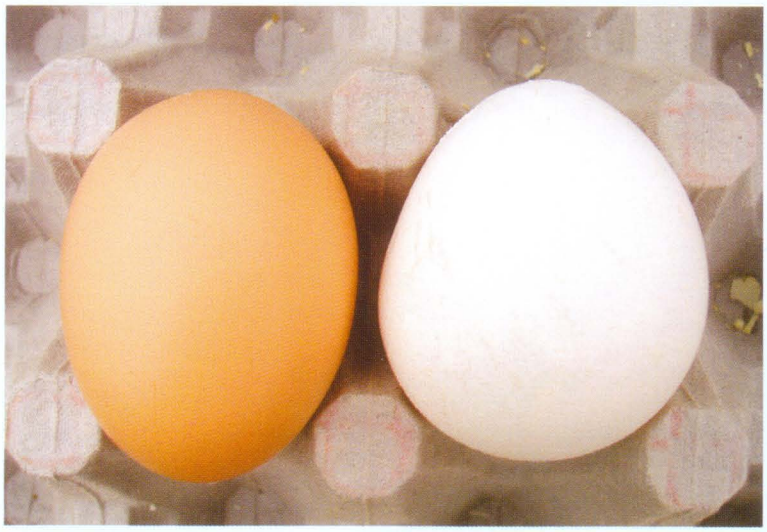
206. Similar inclusion bodies could be sometimes found out in lamina propria of the intestinal mucosa.

207, 208.
The staining of peripheral blood smears reveals a picture, characteristic for septicaemia. The typical gross lesions and the history of the disease allow making a tentative **diagnosis** for HE. The histological detection of intranuclear inclusion bodies in the reticuloendothelial cells of the spleen or intestines confirms the diagnosis. For identification of the HEV, the agar gel precipitation method could be used. The viral antigen could be detected in fresh or frozen spleen tissue, diluted 1:1 with saline or in sera obtained from diseased turkeys. HE should be distinguished from other cases of severe enteritis in turkeys, acute E. coli septicaemia and some other septicaemic states (streptococcosis, fowl cholera etc.). Within weeks after the beginning of the disease, an appropriate antibiotic therapy should be initiated for **prevention** of secondary E. coli septicaemia. Practically, there is no effective therapy for HE in turkeys.



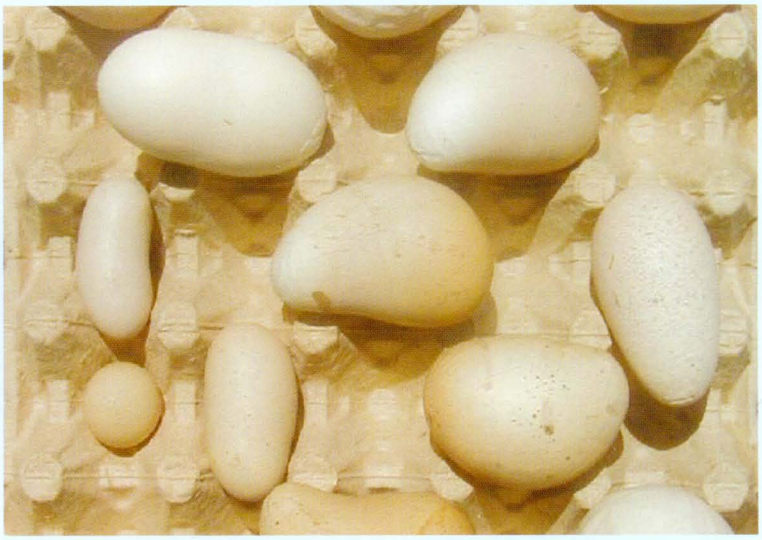


EGG DROP SYNDROME - 1976



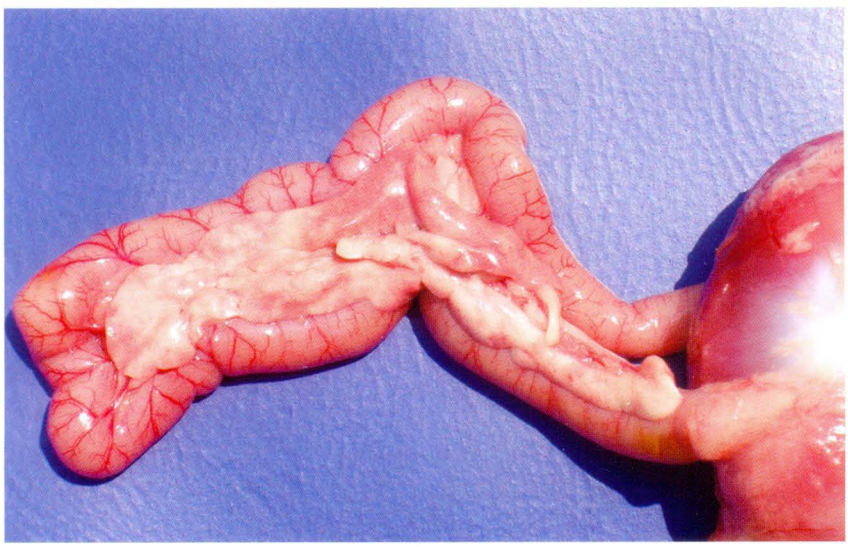
209, 210, 211.
The egg drop syndrome - 1976 (EDS 76) is an infectious disease in layer hens manifested by a quick drop in egg production, failure to reach peak production, irregularly shaped eggs, soft-shelled or shell-less eggs and depigmentation. The aetiological agent is an adenovirus of group III. The horizontal transmission occurs slowly in battery systems and rapidly in floor housing systems.

The first sign is the loss of egg pigmentation, rapidly followed by the appearance of soft-shelled, shell-less of deformed-shell eggs. If defective eggs are discarded, the remaining ones have no problem with fertilization and hatching. The drop could be sudden or prolonged. Usually, it lasts for 4-10 weeks and the egg production is reduced by about 40%. Apart the inactive ovaries and oviduct atrophy, other lesions are not discovered. The replication of the virus in epithelial cells of oviduct glands results in severe inflammatory and dystrophic changes in the mucous coat. The appearance of eggs with impaired quality and the dropped egg production are suggestive for EDS 76. The **diagnosis** is supported by some serological studies and is confirmed after isolation and identification of the virus. In many instances, no antibodies are detected in infected flocks until egg production approaches levels between 50% and peak production.



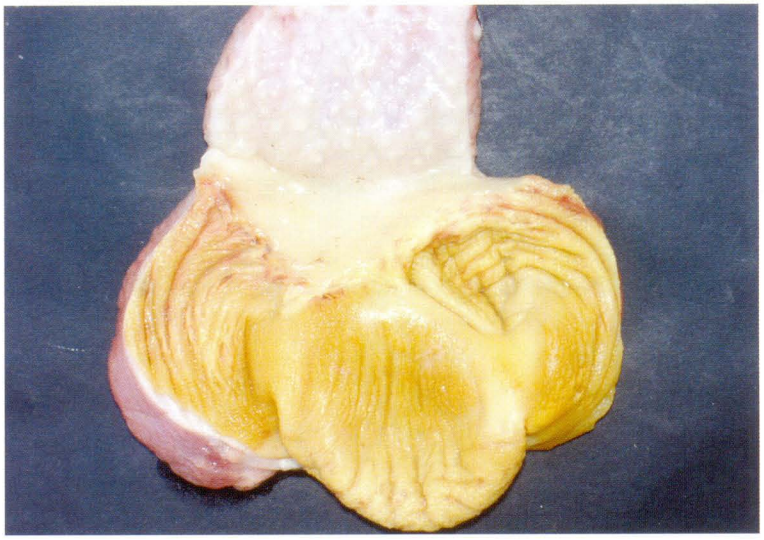
ASSOCIATED ADENOVIRUS INFECTIONS

ADENOVIRUS GROUP I - ASSOCIATED INFECTIONS

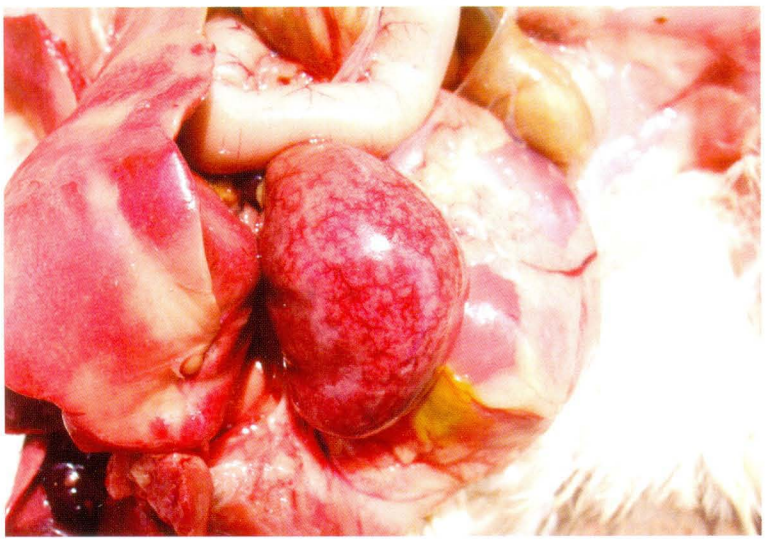


212. Necrotic pancreatitis in broilers. It is characterized by focal necroses in the pancreas.

213. Gizzard erosions in broilers. They are manifested by erosions affecting the cuticle and the underlying tissues of the gizzard.

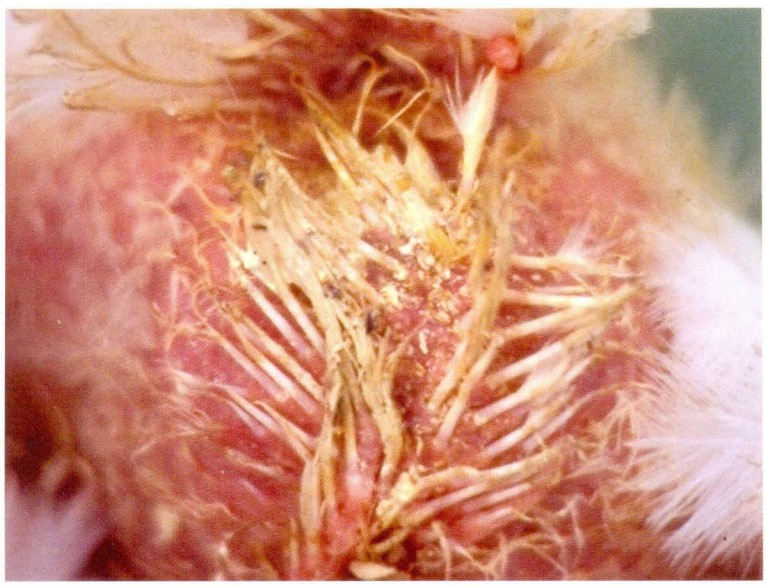


ADENOVIRUS GROUP II - ASSOCIATED INFECTIONS



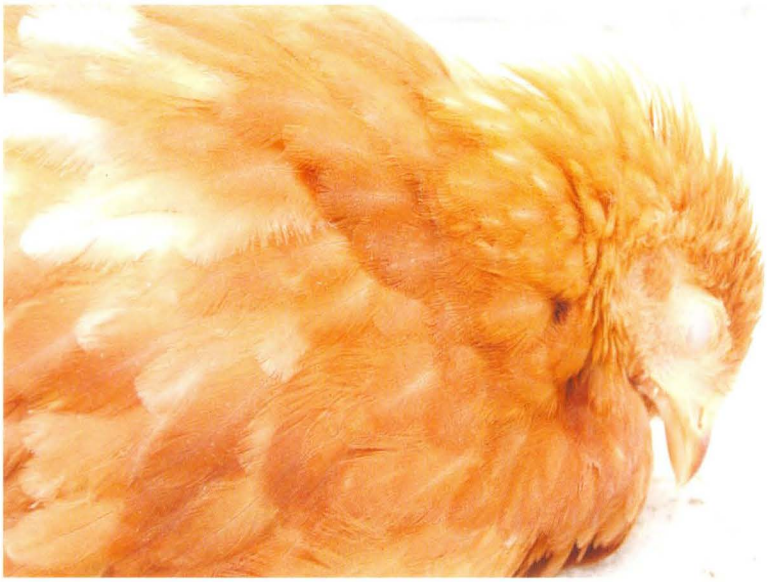
214. Avian adenovirus splenomegaly. Avian adenovirus splenomegaly is characterized by enlargement of the spleen, pulmonary oedema and congestion. It is observed in broiler breeders at the age of 20-45 weeks. Its course is peracute or acute. The mortality could reach 8 - 9%. The most typical lesions are splenomegaly, mottled or marble-like appearance of the spleen, oedema or hyperaemia of lungs.

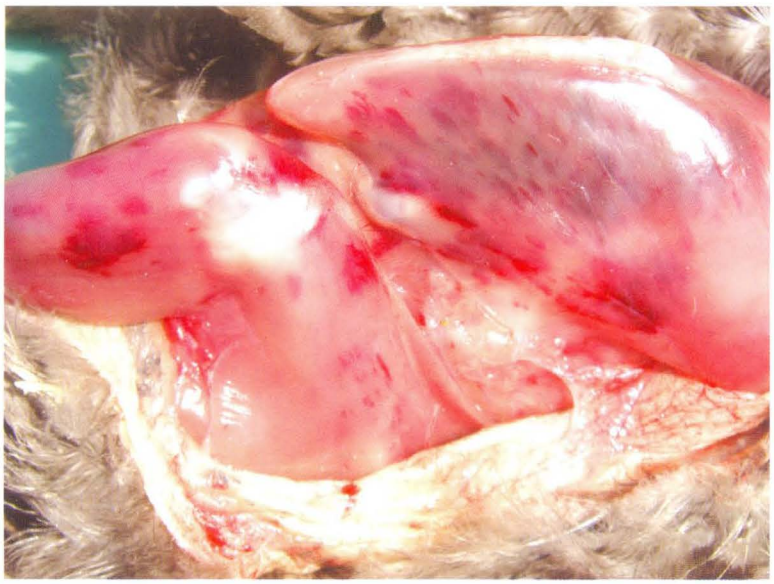
INFECTIOUS BURSAL DISEASE (GUMBORO)



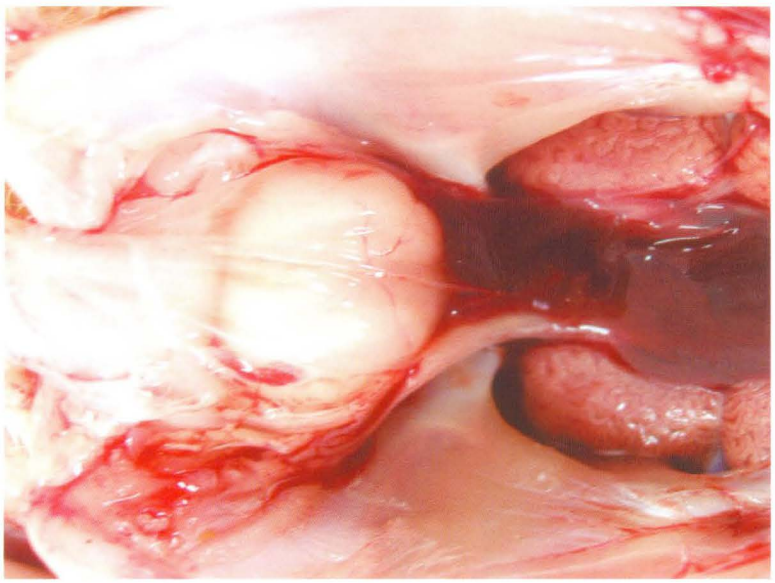
215. Infectious bursal disease (IBD, Gumboro) is an acute, highly contagious viral infection in chickens manifested by inflammation and subsequent atrophy of the bursa of Fabricius, various degrees of nephroso-nephritis and immunosuppression. Clinically the disease is seen only in chickens older than 3 weeks. The feathers around the vent are usually stained with faeces containing plenty of urates.

216. The period of most apparent clinical symptoms and high death rate is at the age of 3 - 6 weeks. IBD could however be observed as long as chickens have a functioning bursa (up to the age of 16 weeks). In chickens younger than 3 weeks, IBD could be subclinical, but injured bursa leads to immunosuppression. Also, diarrhoea, anorexia, depression, ruffled feathers, especially in the region of the head and the neck are present.

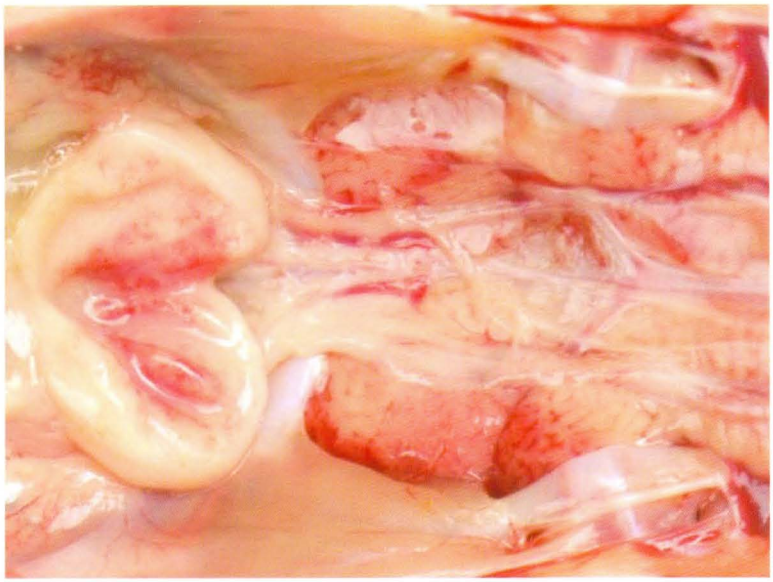




217. A natural IBD infection is mostly observed in chickens. In turkeys and ducks it could occur subclinically, without immunosuppression. Most isolates of the IBD virus in turkeys are serologically different from those in chickens. In premises, once contaminated with the IBD virus, the disease tends to recur, usually as subclinical infection. The dead bodies are dehydrated, often with haemorrhages in the pectoral, thigh and abdominal muscles.

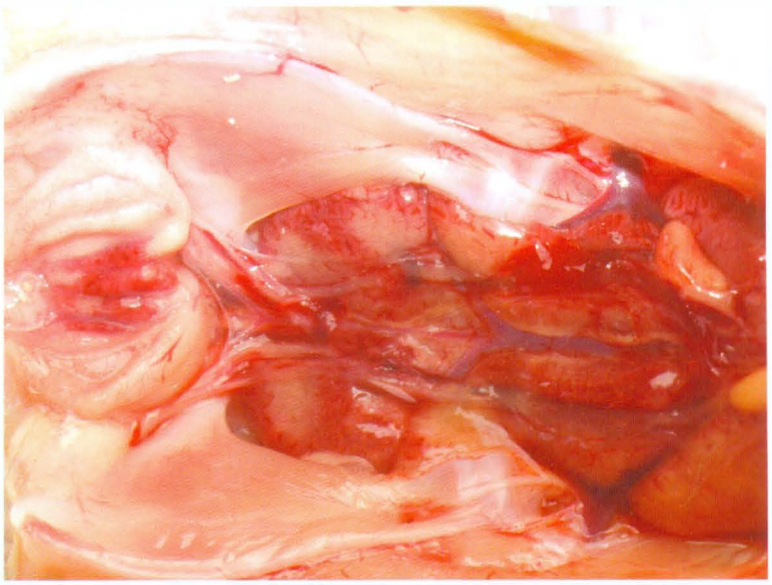


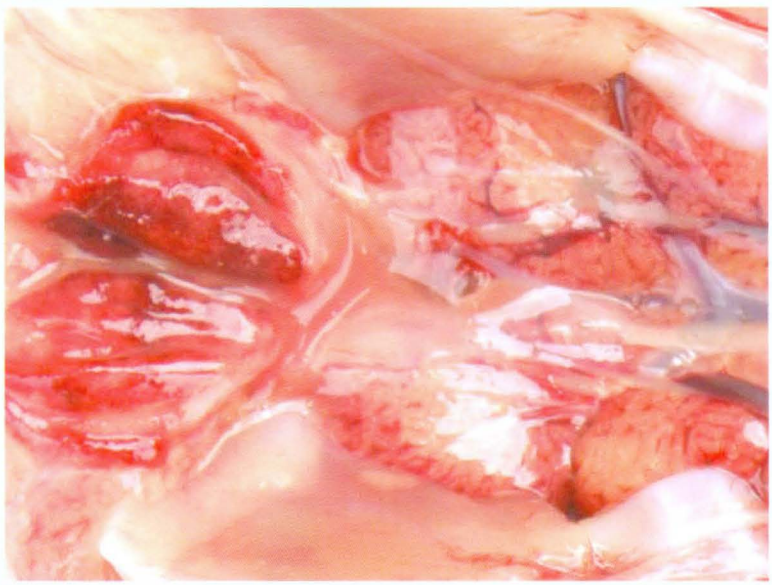
218. The IBD virus belongs to the Birnaviridae family of RNA viruses. Two serotypes are known to exist, but only serotype 1 is pathogenic. The virus is highly resistant to most disinfectants and environmental onditions. In contaminated premises, it could persist for months and in water, forage and faeces for weeks. The incubation period is short and the first symptoms appear 2 - 3 days after infection. The lesions in the bursa of Fabricius are progressive. In the beginning, the bursa is enlarged, oedematous and covered with a gelatinous transudate.

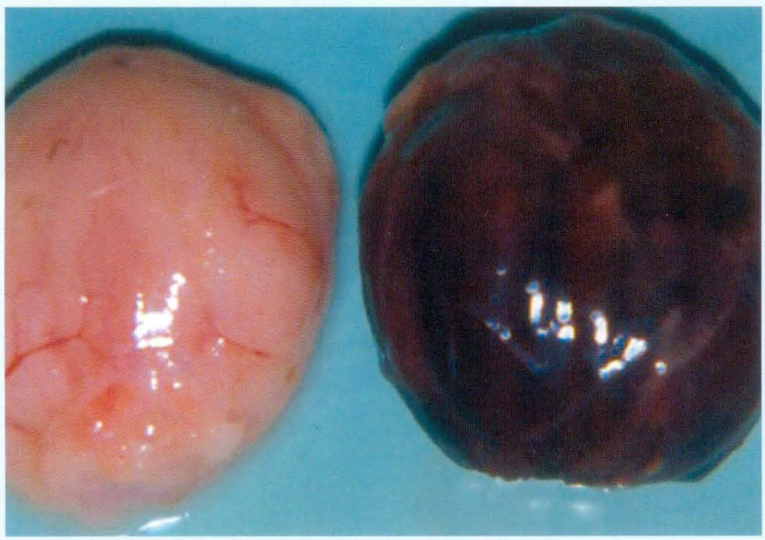


219. The IBD virus has a lymphocidic effect and the most severe injuries are in the lymph follicles of the bursa of Fabricius. Most commonly, IBD begins as a serous bursitis.

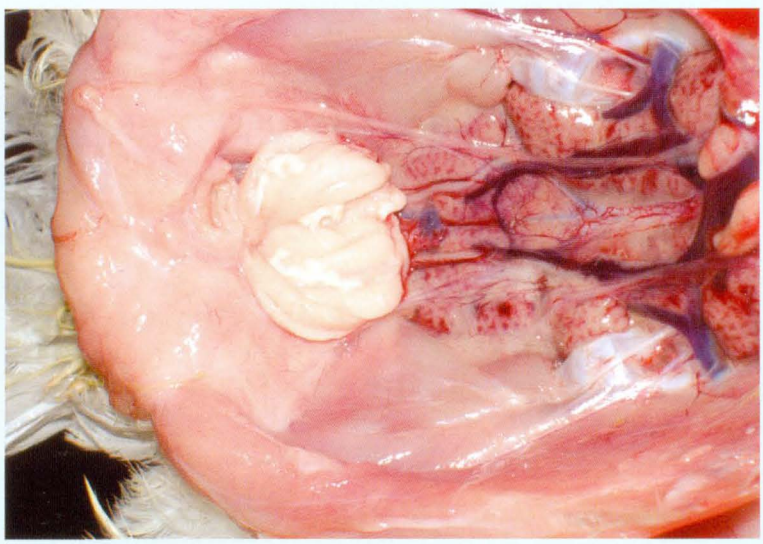
220, 221, 222. IBD lesions undergo various stages of serous haemorrhagic to severe haemorrhagic inflammation. The morbidity rate is very high and could reach 100%, whereas the mortality rate: 20 - 30%. The course of the disease is 5 - 7 days and the peak mortality occurs in the middle of this period.

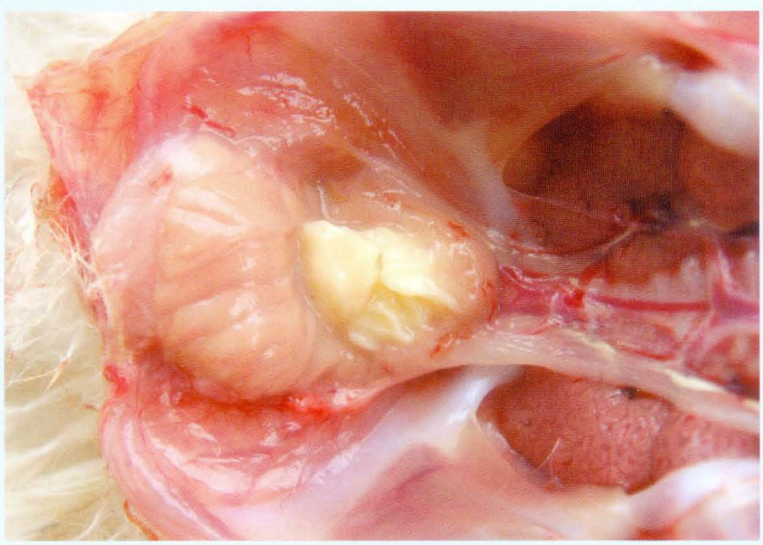


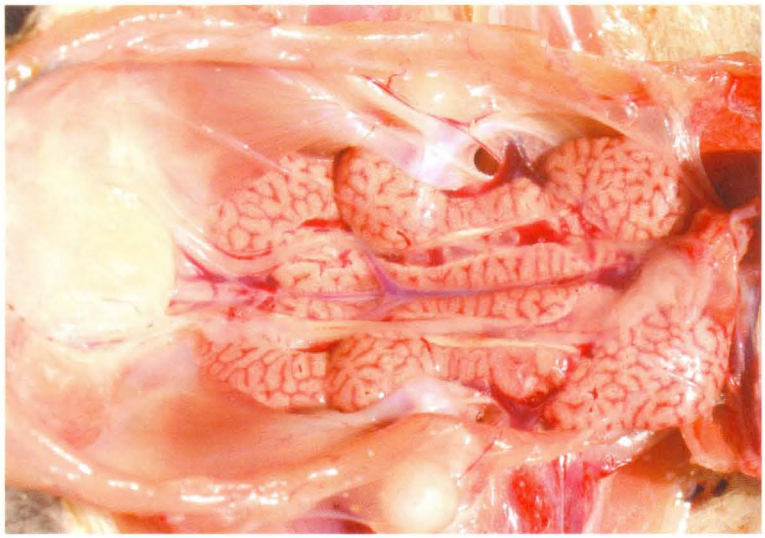




223, 224. In some cases, the bursa is filled with coagulated fibrinous exudate that usually forms casts with the shape of mucosal folds. In birds surviving the acute stage of the disease, the bursa is progressively atrophying. Microscopically, an atrophy of follicles into the bursa is observed secondary to inflammatory and dystrophic necrobiotic alterations.

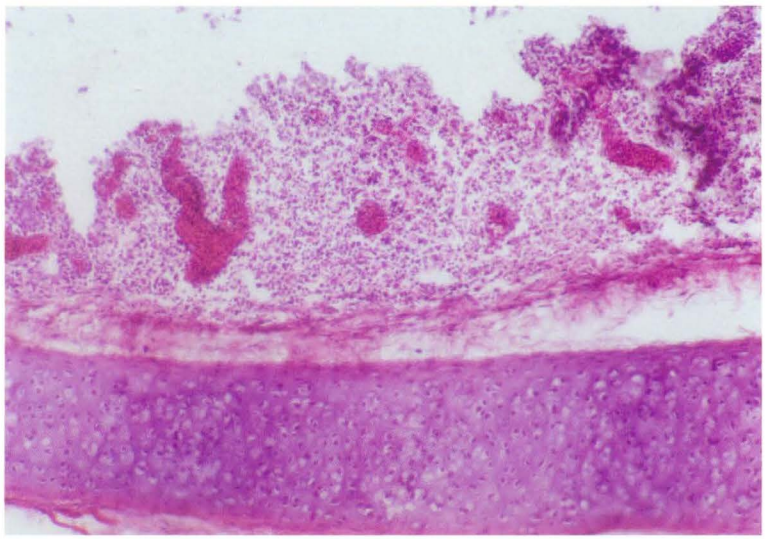






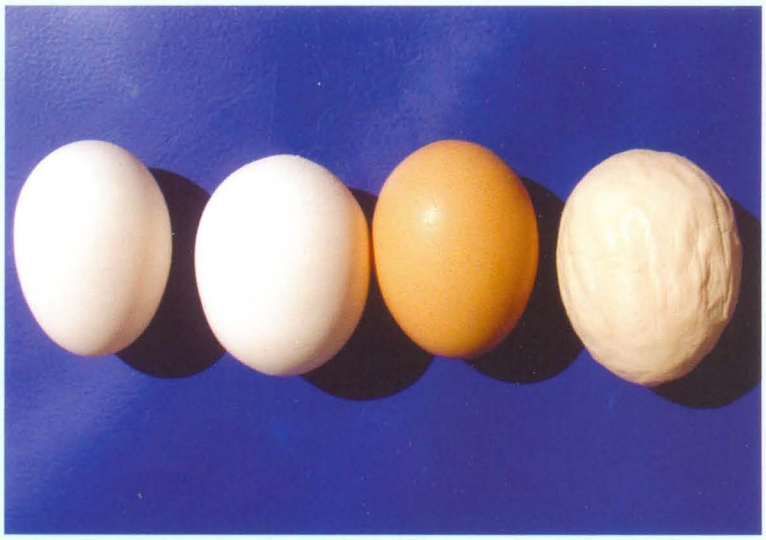
225. The kidneys are affected by a severe urate diathesis. In an acute outbreak and manifestation of the typical clinical signs, the diagnostics is not difficult. The **diagnosis** could be confirmed by detection of typical gross lesions throughout a patho-anatomical study. IBD should be differentiated from IBH (inclusion body hepatitis). The application of live vaccines in chickens is a key point in the **prevention** of IBD and should be related to the levels of maternal antibodies.

INFECTIOUS BRONCHITIS (IB)



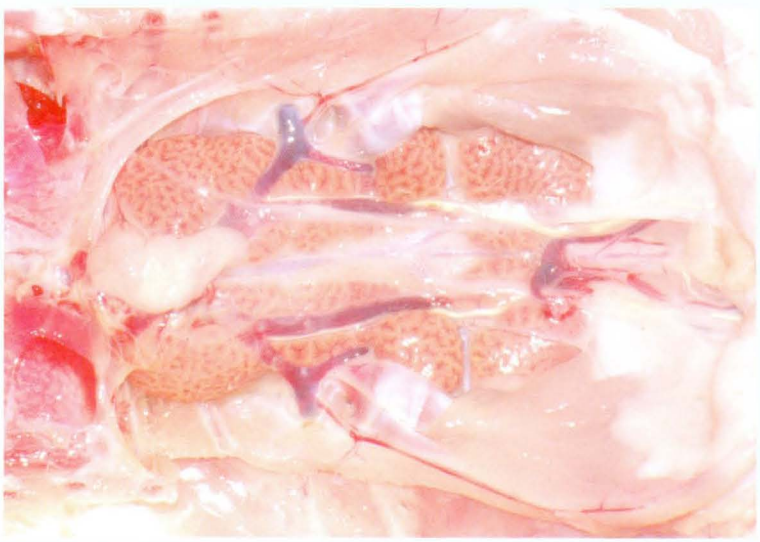
226. In chickens up to the age of 4 weeks, IB is manifested by severe respiratory signs (sneezing, coughing, and rales). Rhinites and conjunctivites, depression and crowding around heat sources are observed. The death rate could reach 100%. The mortality in young chickens is usually insignificant provided that a secondary infection with a different agent is not occurring. In such cases, there is a moderate to severe inflammatory cell infiltration of upper respiratory tract mucosa, resulting in thickened and more compact mucosa.

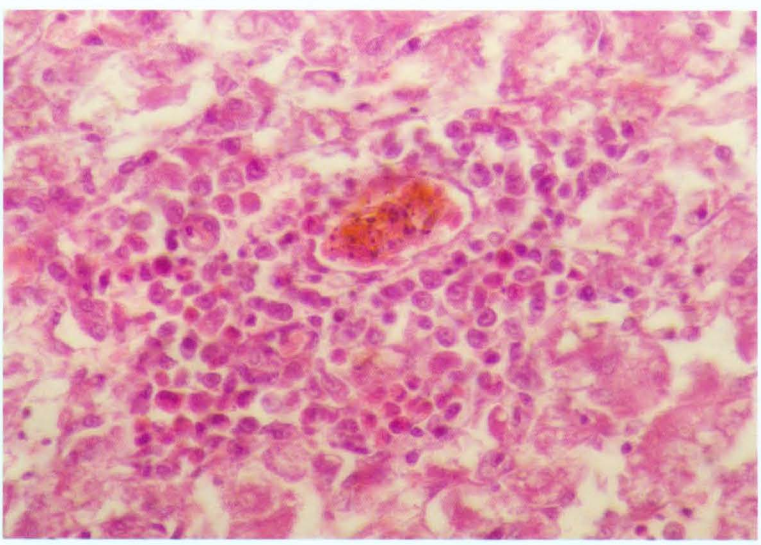
227, 228. In layer hens infected with the IB virus, oophorites and dystrophic necrobiotic lesions affecting primarily the middle and the last thirds of oviduct's mucous coat are observed. The consequences are drop in egg production, appearance and increase in the number of deformed and pigmentless eggs or eggs with soft shells and watery egg white.

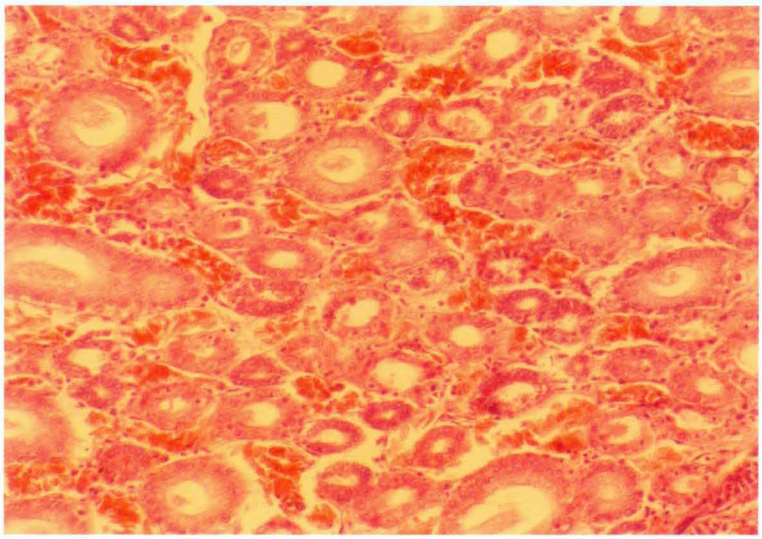


The oviduct is atrophied, cystic, with deposits of yolks or completely formed eggs in the abdominal cavity (the so-called internal layer). IB is caused by a coronavirus. It is characterized by a rich antigenic diversity and that is why many serotypes (Massachusetts, Arkansas 99, Connecticut, O72 etc.) are identified. Often, the infection's course is complicated with the involvement of *E. coli*, *M. gallisepticum*, the laryngotracheitis virus etc.

229, 230, 231.
The nephrotropic strains of the IB virus cause severe inflammatory and dystrophic necrobiotic damages of kidneys: urolithiasis (229), interstitial nephritis (230), haemorrhages (231) that considerably increase the death rate. Under natural conditions, only hens are infected. Non-immune birds of all ages are susceptible. The disease is even seen in vaccinated flocks. The serological methods (VN, ELISA etc.) are widely used in the **diagnostics**. At present, PCR is used for rapid identification of IB virus serotypes. IB should be distinguished from other acute respiratory disease as ND, laryngotracheitis and infectious coryza. The vaccination with live or killed vaccines is effective only if they contain the respective serotype of the virus for the given region.



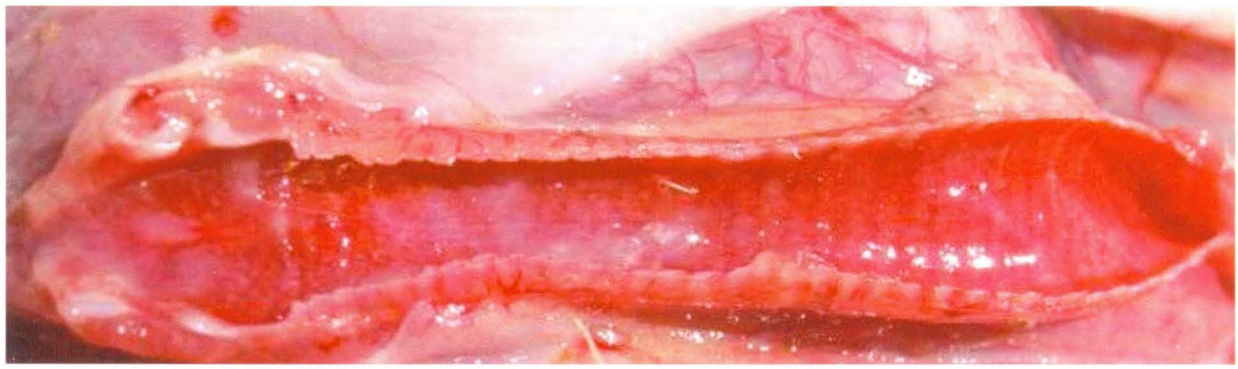




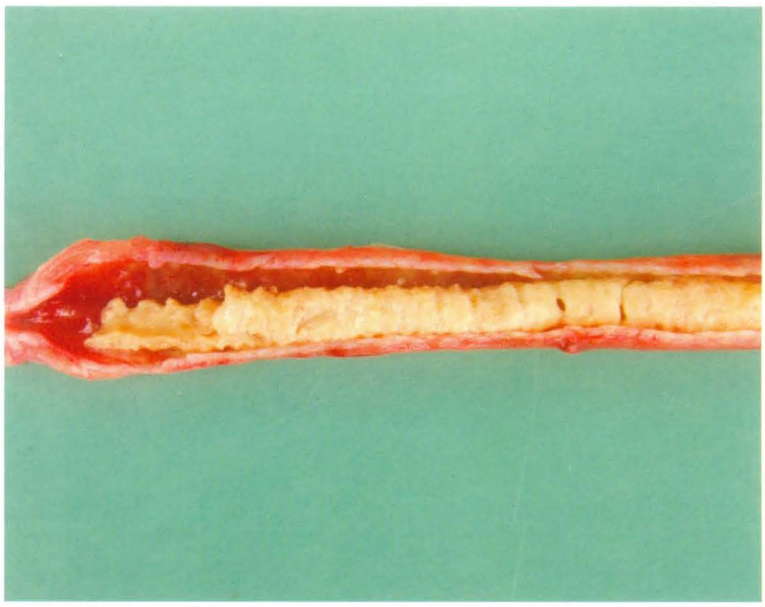
LARYNGOTRACHEITIS

232. Laryngotracheitis (LT) is a viral infection in hens, pheasants and peacocks characterized by catarrhal haemorrhagic to fibrinous inflammation of the respiratory tract. It is manifested in laryngotracheal and conjunctival form. In the laryngotracheal form, suffocation, rales and cough are observed. The head and the neck are strongly extended forward and upward during inspiration.

233. The mucous coats of the larynx and the trachea are catarrhally haemorrhagically to fibrinously inflamed. Most outbreaks are encountered between the age of 4 and 14 weeks although the disease affects fowl of any age. LT is caused by a herpesvirus that is relatively resistant.



234. Haemorrhagic laryngotracheitis. The morbidity rate of LT reaches 50-70% and the death rate: 10-20%. Often, it goes on as a complicated infection after the involvement of *E. coli*, *St. aureus*, *M. gallisepticum* etc.



235. In some cases, casts of haemorrhagic or fibrinous exudate are formed that could almost completely obturate the larynx and the trachea. Source of the infection are sick and convalescent birds, the latter being prolonged carriers of the virus (up to 1 - 2 years). With this regard, a certain stationarity is observed.

236. In the conjunctival form of LT, wet eyes, tear secretion and oedema of infraorbital sinuses are observed, especially in a complicated infection. The typical clinical and morphological signs are sufficient to assume the presence of LT. The **diagnosis** is confirmed with the detection of intranuclear inclusion bodies in the trachea throughout the histological study in the early stages of the disease, serological studies (VN, ELISA) etc. LT should be differentiated from IB, SHS, *M. synoviae* infections etc. Premises, contamined with the LT virus, should be freed, cleansed, disinfected and occupied again after 5 - 6 weeks. The vaccination of unaffected birds and these from other premises of the infected farm could protect and stop subsequent outbreaks.

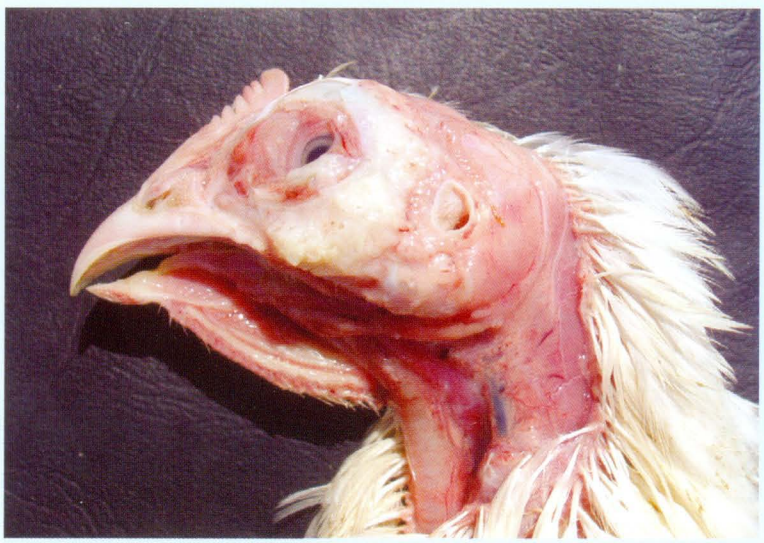
SWOLLEN HEAD SYNDROME

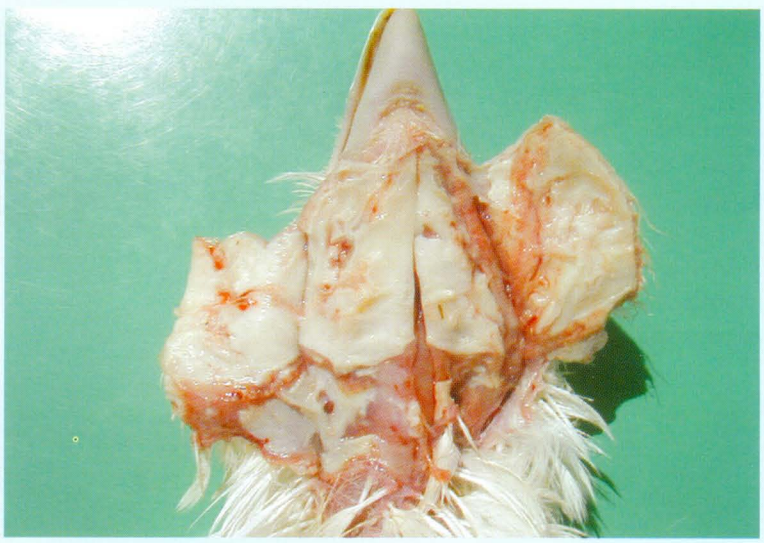
237, 238.
Swollen head syndrome (SHS) is a complicated infection in broilers and broiler breeders, where the primary aetiological agent is an avian pneumovirus (APV) and the secondary - usually *E. coli*; it is characterized by respiratory and nervous signs. In broilers chickens, SHS is generally seen after the 4th week of life. The first clinical signs are sneezing, coughing, rales and conjunctivites. A profuse tear secretion, reddened conjunctivas and a characteristic oblong almond-like shape of eyes are observed. The inflammatory exudate is initially transparent, but afterwards becomes opaque.

239, 240. Subcutaneous oedemas in the head region, involving unilaterally or bilaterally the periorbital sinuses and the mandibular space, are emerging. A seasonal pattern in the prevalence with peaks during spring and summer is observed.

The virus is transmitted by direct contact of infected and susceptible birds. The airborne route of transmission from farm to farm is widely distributed. The principal predisposing cause is the poor microclimate, bad ventilation, high dust and ammonia levels etc.

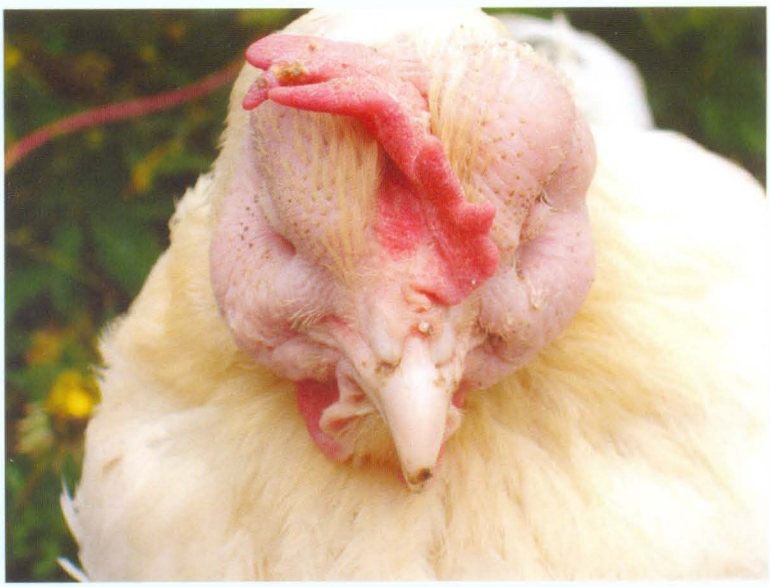
241, 242. After removal of the covering skin, deposits of serofibrinous exidate are observed. APV is a virus from the Paramyxoviridae family. The pneumovirus is present in respiratory secretions and discharges. It survives for a long time in cold and wet environment. *E. coli* follows the APV infection of the upper respiratory tract. The triggered inflammatory response results in accumulation of exudate in the subcutaneous tissue. In many instances, a croupous pneumonia develops at a later stage consequently to contamination with other pathogens.





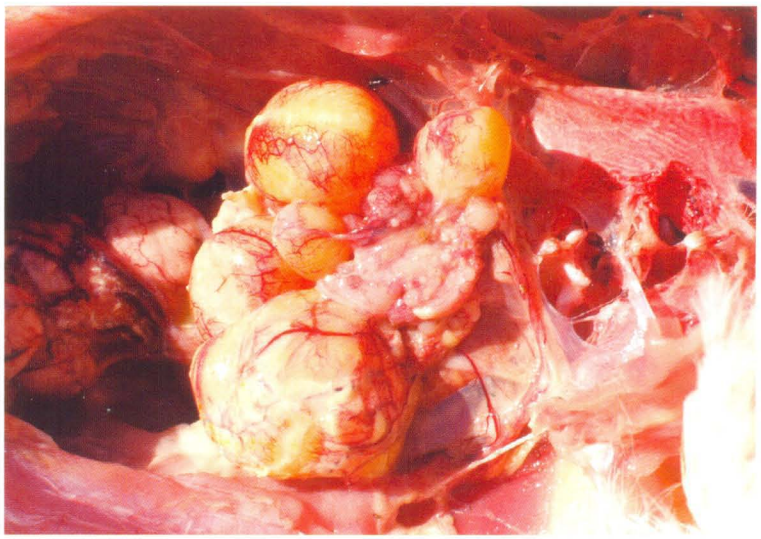
243, 244, 245.

SHS in broiler breeders is usually encountered around or after the peak egg laying period only in female birds. Unilateral or bilateral swellings of the head, affecting the periorbital sinuses, the mandibular space and the wattles are seen. The conjunctiva and the mucous membranes of sinuses are considered to be the entrance door of the infection. A unique cytotoxin has been identified in many *E. coli* isolates in SHS that could be involved in the pathogenesis of the disease. The inflamed by the virus infection conjunctiva-associated lymphoid tissue is the site where the bacterium enters the subcutaneous tissue.

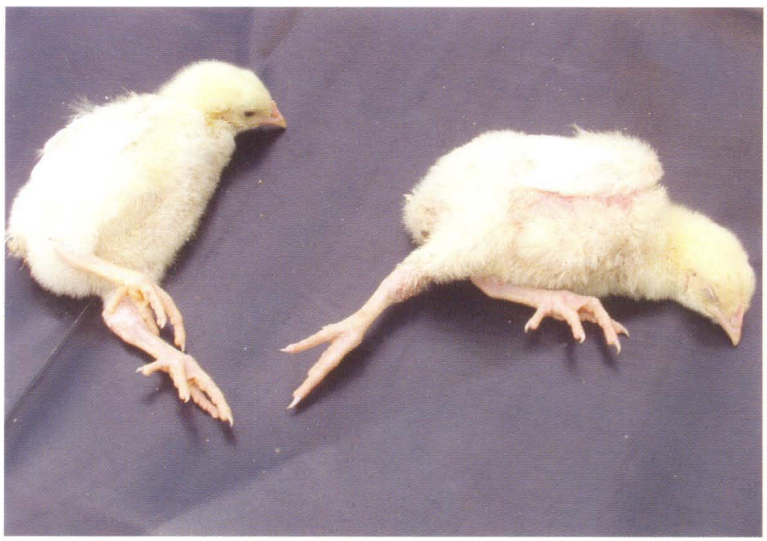


246. Frequently, nervous signs are observed in broiler breeders (opisthotonus, torticolis) due to inflammatory processes in pneumatic skull bones and the middle ear.

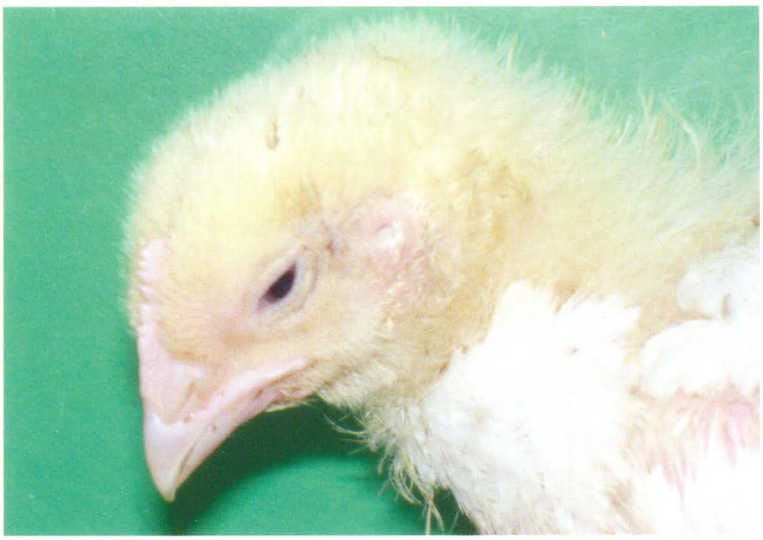
247. In laying hens, the ovaries are affected in many instances (sero-fibrinous oophoritis), resulting in reduced egg production. The **diagnosis** is based upon the distinctive clinico-morphological signs. SHS should be differentiated from *Mycoplasma* and *Pasteurella* infections and the skin form of aspergillosis. **Prevention** - improvement of the microclimate of premises, use of live and killed vaccines.



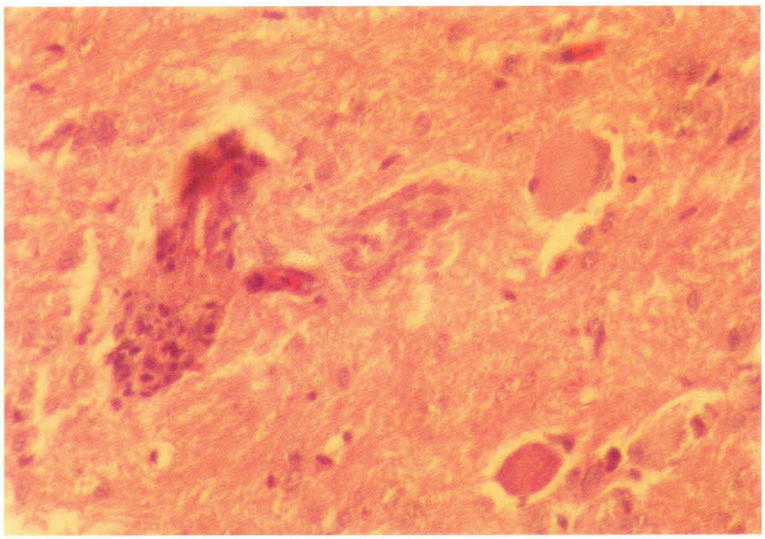
INFECTIOUS ENCEPHALOMYELITIS



248. The infectious encephalomyelitis (IEM) is characterized by signs of ataxia, progressing to paralysis, prostration and marked tremor of the head and the neck, and because of that, is also called epidemic tremor. The chickens with prostration are usually in lateral recumbency.

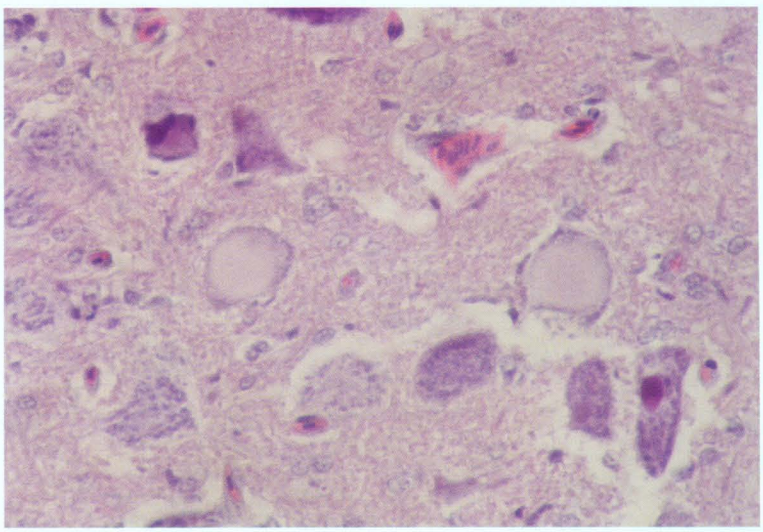


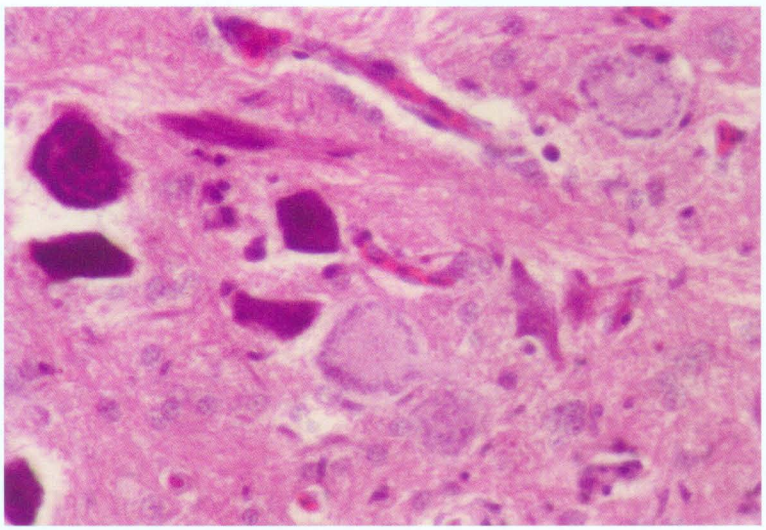
249. The tremor could be unapparent, but is often perceptible when the chicken is held gently with the hand and carefully looked at. The expression of the eyes is dull. IEM outbreaks are generally observed in chickens at the age between 8 - 9 and 20 days. The morbidity rate could reach 40 - 60%. The average death rate is about 25%, but could be more than 50%. Gross lesions are not present.



250. The histological lesions are specific and with a diagnostic value. A nonpurulent encephalomyelitis with marked perivascular clusters is present. The IEM virus is from the Picornaviridae family. The previous studies placed the virus in the Enterovirus genus, but based on the most recent studies, it is provisionally referred to the Hepatovirus genus. The virus is found in faeces of infected chickens and could survive there for at least 4 weeks. It is relatively highly resistant to environmental conditions.

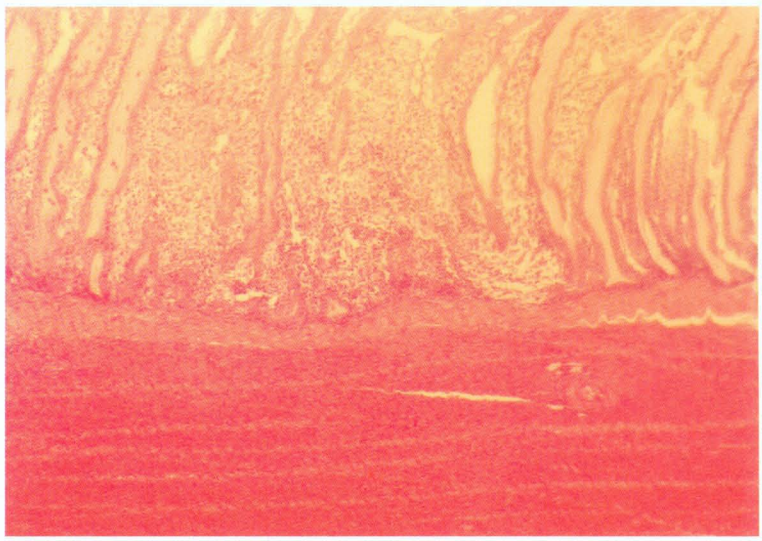
251, 252. A particularly valuable finding is the central chromatolysis (251) of neurons in segments from the lumbosacral widening of the spinal cord and more rarely, chromatopyknosis (252). The infection occurs in non-vaccinated broiler breeder flocks and its course is subclinical. A vertical transmission of the infection to susceptible birds is realized by the eggs laid during this period.

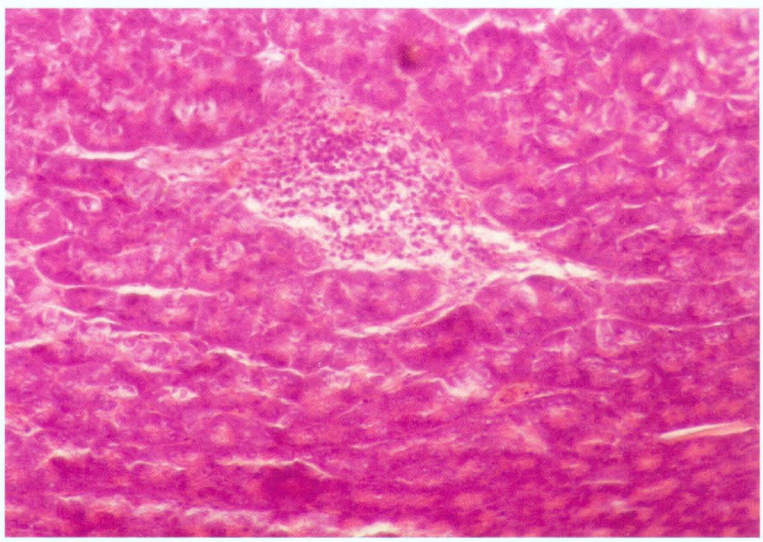




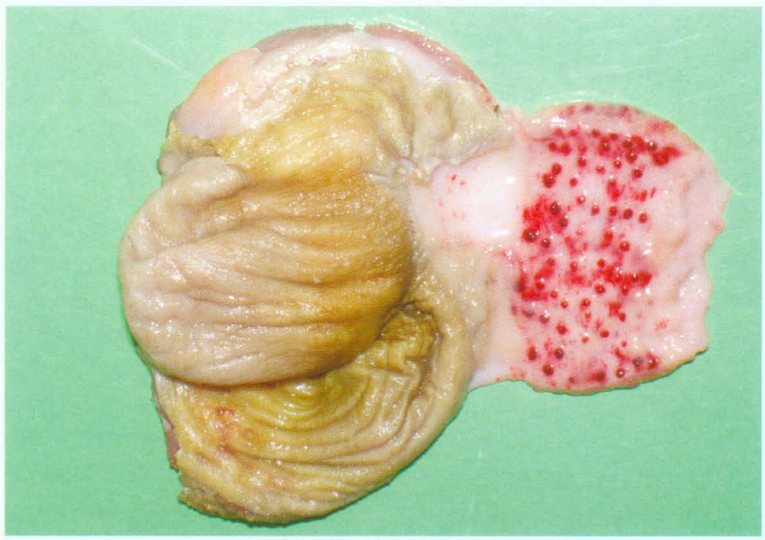
At the same time, parents acquire immunity and transmit it to the next generations thus protecting them from IEM. So, clinical manifestation of IEM is observed in chickens, hatched from the eggs during the 2- or 3 week period after breeder flock's infection. The next generations are immune.

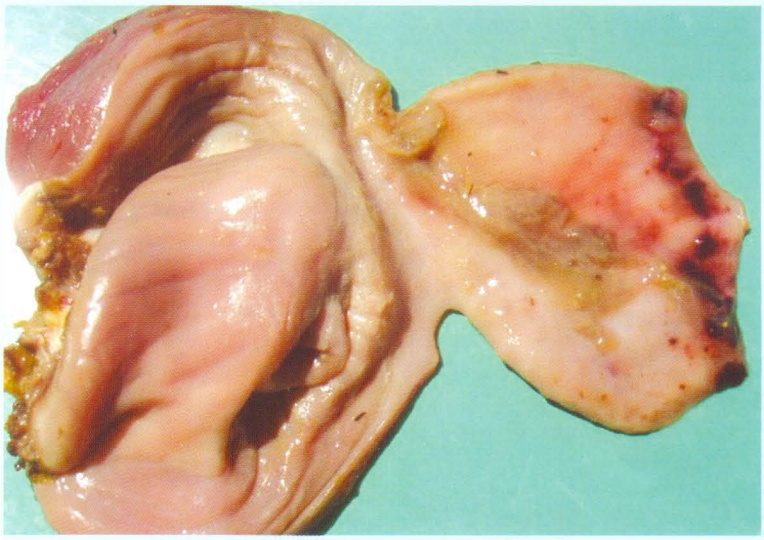
253, 254. Valuable diagnostic findings are the dense lymphoid clusters in the muscles of the proventriculus and the gizzard (253) and in the pancreatic interstitium (254). The history of the disease, the age of onset and the typical nervous signs, especially the head tremor are indicative for the **diagnosis** that could be finally confirmed by histology. IEM should be differentiated from other diseases with nervous symptoms as encephalomalacia, mycotic encephalites, toxicoses (salt, pesticides). **Prevention** - vaccination of breeder flocks with regard to ensuring maximum protection of their offspring. The chickens from naturally infected flocks obtain an adequate maternal immunity that preserves them from the disease.

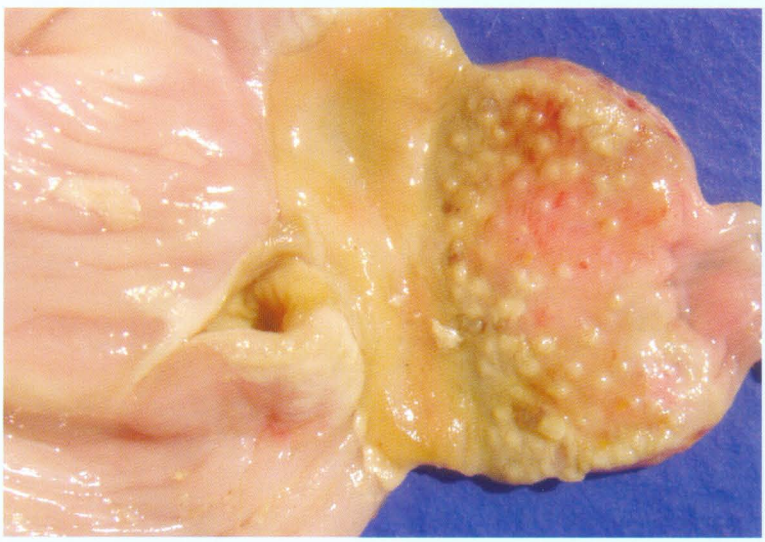




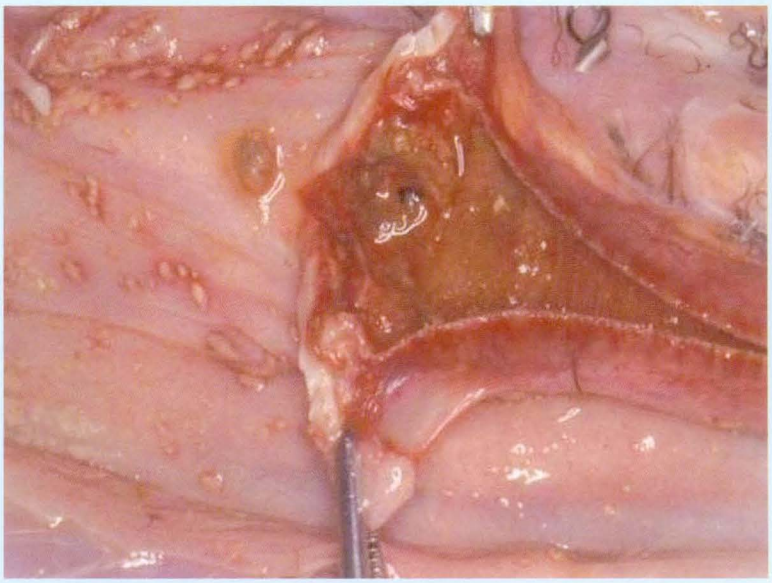
NEWCASTLE DISEASE

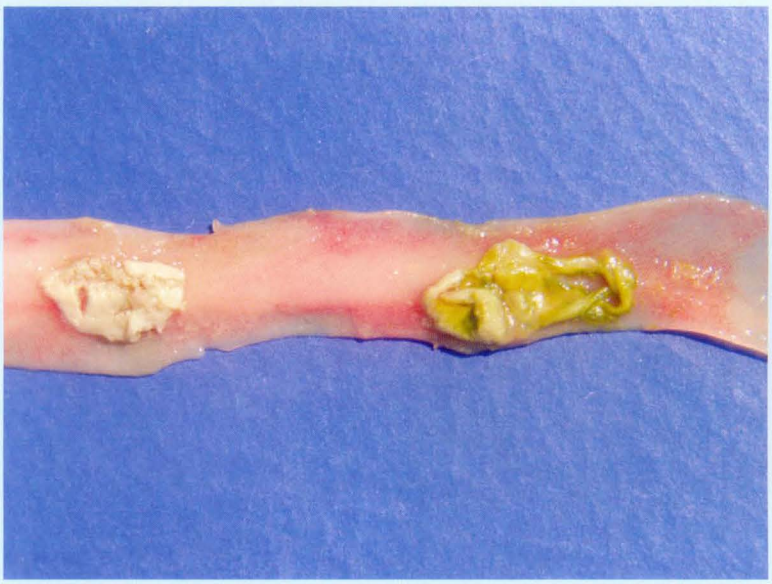


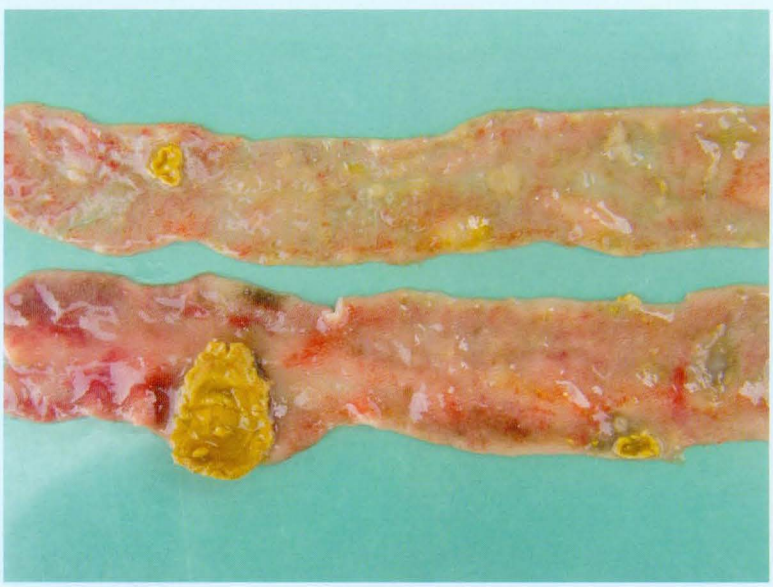




255, 256, 257.
The Newcastle disease (ND) is a highly contagious disease in many species of domestic, exotic and wild birds that, depending on its tropism, is characterized by marked variations in morbidity, death rate, symptoms and lesions. The clinico-morphological signs possess a distinct viscerotropic or neurotropic character. In the **viscerotropic form**, haemorrhagic diphtheritic lesions of the entire alimentary tract, from the beak to the vent, are present. The haemorrhages of gizzard epithelium are remarkable. The mucous coat is oedematous, covered with thick mucus and mottled with haemorrhages varying from single to multiple, sometimes gathered at the boundaries with the gizzard or the oesophagus.

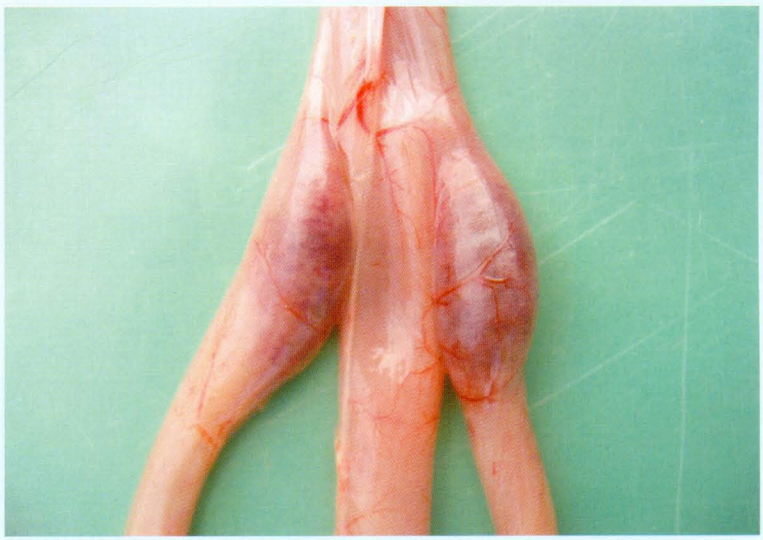


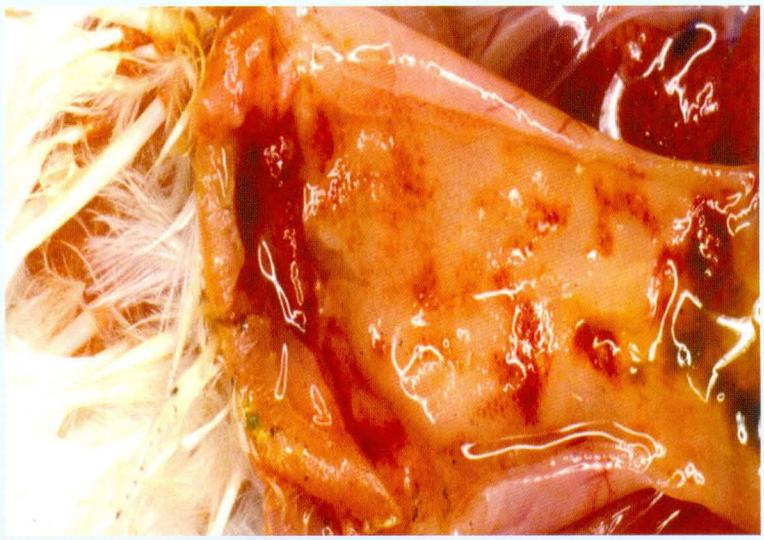


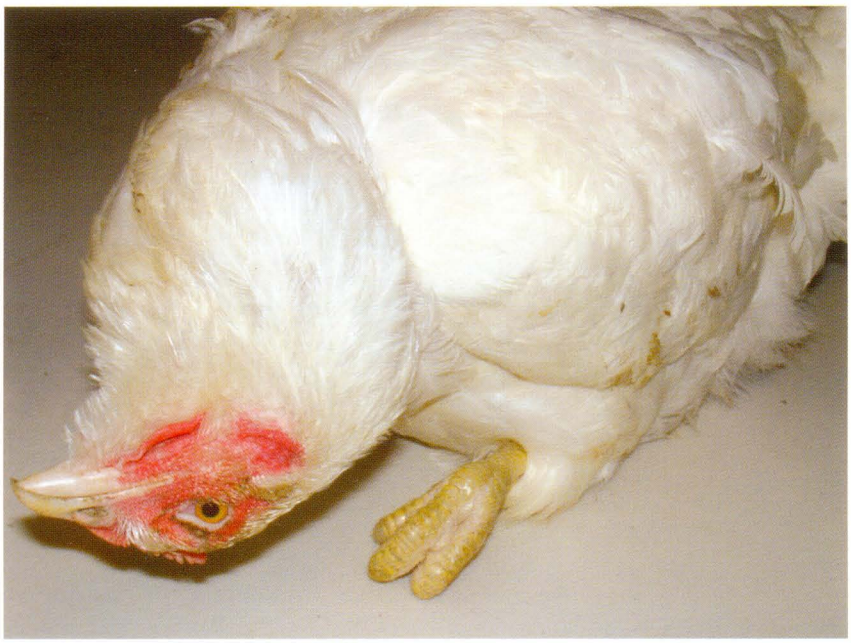


258, 259, 260.
Typical for this form are the haemorrhagic necrotic and focal diphtheroid lesions affecting the mucosa of the buccal cavity (258), the stomach and the intestines (259, 260). The disease is generally prevalent in hens, more rarely in turkeys, exotic or wild birds. Birds at any age are susceptible. It is caused by a paramyxovirus. Depending on their pathogenicity, the numerous known strains are classified as lentogenic, mesogenic and velogenic. The vaccines made of lentogenic strains provoke a shorter immunity that requires a revaccination. The vaccines from mesogenic strains result in a lasting immunity, but could provoke a lethal issue especially in birds without a primary immunity created on the basis of lentogenic vaccinal strains.

261, 262. A frequent finding is the enlargement and haemorrhages of caecal tonsils (261) and haemorrhagic cloacitis (262). Usually, these lesions begin from the lymphoid tissue of the mucous coat. Virus-containing excreta of infected birds, that contaminate the forage, water and the environment, are the source of infection. The infection is transmitted mainly by an oral route, the airborne or contact transmission being more infrequent. The virus, contained in incubated eggs, results in embryo's death and then perishes. There is no permanent carriership of the virus. An important factor in the transmission of velogenic viruses could be the exotic birds and fighting cocks. The death rate could arrive at 70-100%.





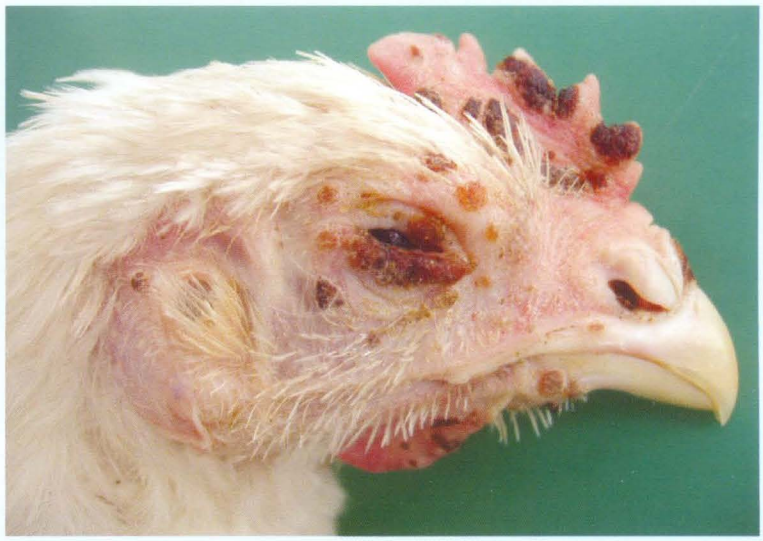


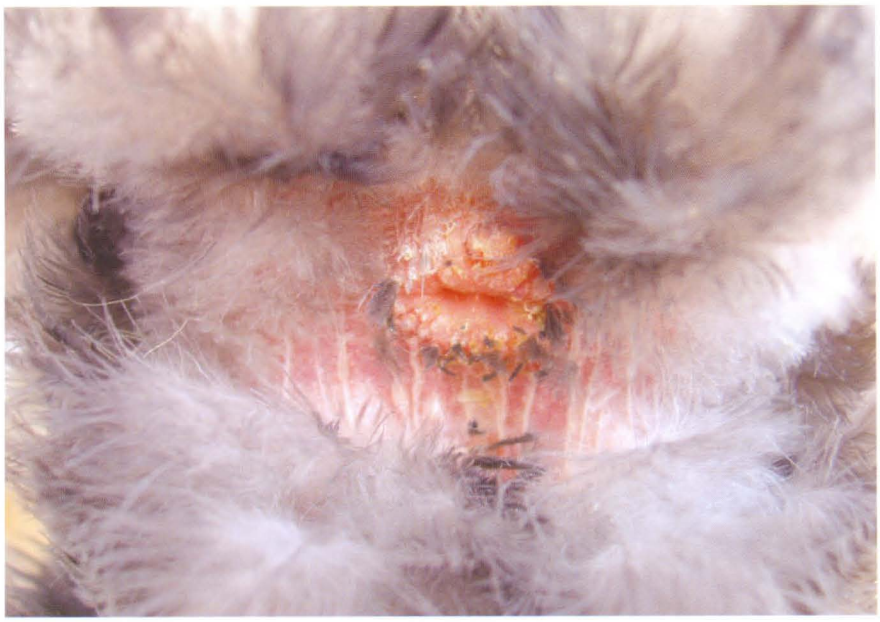
263. The neurotropic form of the disease is clinically manifested by ataxia, opisthotonus, torticolis, paresis and paralysis of legs. This form is frequently accompanied by respiratory symptoms. Histopathologically, the picture of nonpurulent lymphocytic encephalomyelitis is observed.

264. The lesions of paramyxovirosis in pigeons are entirely identical. On the basis of the history and the clinico-morphological signs, a tentative **diagnosis** could be made, but its laboratory confirmation is mandatory. ND should be distinguished by avian influenza, fowl cholera etc.

FOWL POX

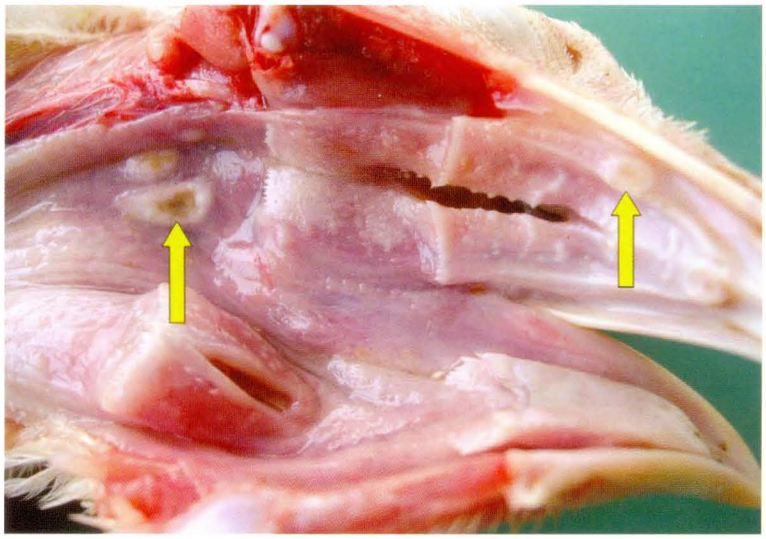
265, 266.
Fowl pox (FP) is a viral disease in hens, turkeys and many other birds, characterized by cutaneous lesions on the featherless skin and/or diphtheritic lesions of mucous coats of the upper alimentary and respiratory tract. FP is encountered in either cutaneous or diphtheritic form or in both. In most outbreaks, the cutaneous form is prevailing. The lesions vary according to the stage of development: papules, vesicles, pustules or crusts. The lesions are usually in the region of the head.





267. FP lesions around the vent in a pigeon.
FP is caused by an epitheliotropic DNA virus from the Avipox genus, the Poxviridae family. Some virus types (strains) exist: fowl pox virus, turkey pox virus, pigeon pox virus, canary pox virus etc., different in pathogenic and immunogenic aspects. The viruses are very resistant to environmental factors and could persist for several months.

268, 269.
Frequently, the conjunctival mucosa, injured by the pox virus, is an entrance door for additional contamination (*E. coli, Staphylococcus spp.* etc.) and development of complications. The infection is mechanically spread by dissemination of the virus through desquamation of crusts that contain it. Some mosquitoes and blood suckling arthropods could also distribute the virus. The mosquitoes remain infective for several weeks. The incubation period is from 4 to 10 days. The disease is spread slowly and many weeks could pass between its emergence and severe outbreaks occurrence.

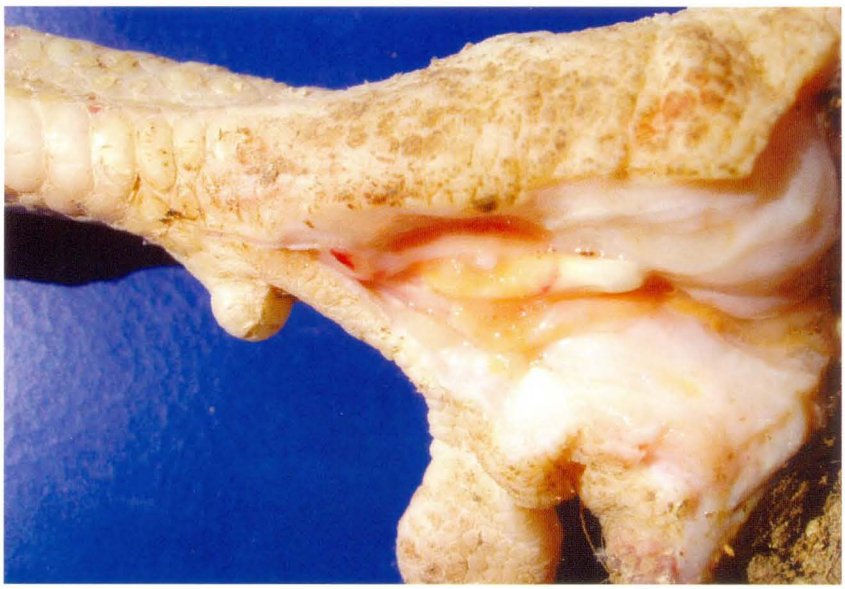


270. Diphtheritic lesions look like whitish or yellowish plaques that are deposited and grown on the mucous coats of the buccal and nasal cavities, the sinuses, the larynx, the pharynx, the trachea or the oesophagus (arrows). The **diagnosis** is made upon the typical cutaneous and diphtheritic lesions. The **prevention** is performed by vaccinations that could be made at any age, if necessary.

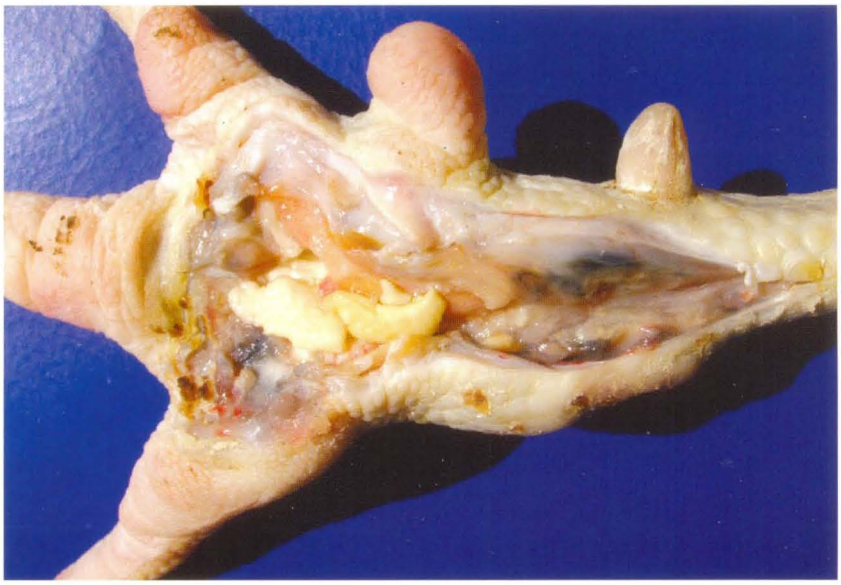
REOVIRUS INFECTIONS

271. Viral arthritis/tenosynovitis.
The most frequent reovirus-associated disease in poultry is viral arthritis. Clinically it is manifested by lameness and swellings affecting primarily tarsometatarsal joints and the feet. Many infected birds are in a good general condition, but some could be lethargic and exhausted. The mortality is very low. The infections affect predominantly meat type poultry.

272. In some cases, joint cavities or tendon sheaths contain a small amount of straw-yellow exudate whereas in other the exudate is haemorrhagic or fibrinous.

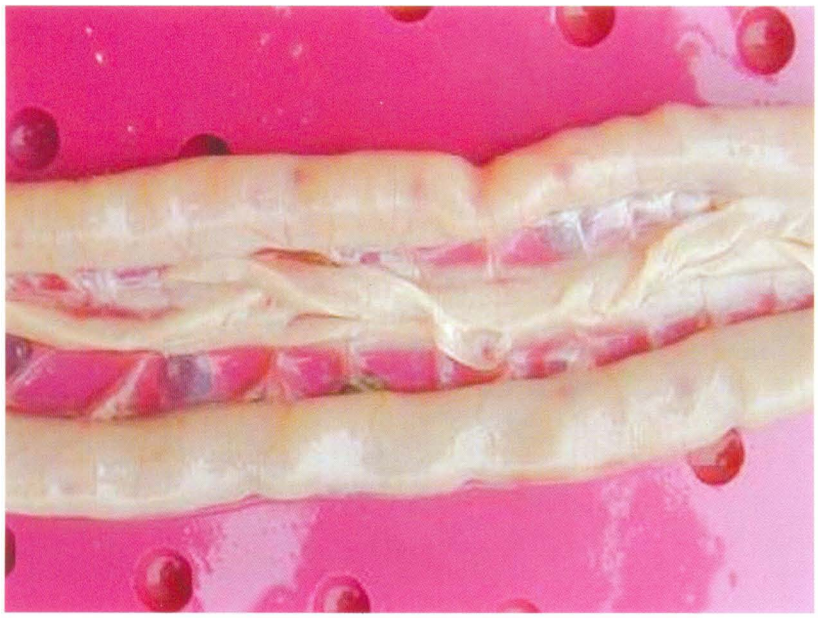


273. Fibrinous tenosynovitis. The inflammation of the tendon progresses to a chronic type lesion characterized by tissue fibrosis in the affected area. The aetiological agent is a reovirus, member of the *Orthoreovirus* genus, Reoviridae family. Several serotypes are identified. For their identification, the agar gel precipitation test could be used. Reoviruses are highly resistant to a number of environmental factors (temperature, pH etc.).



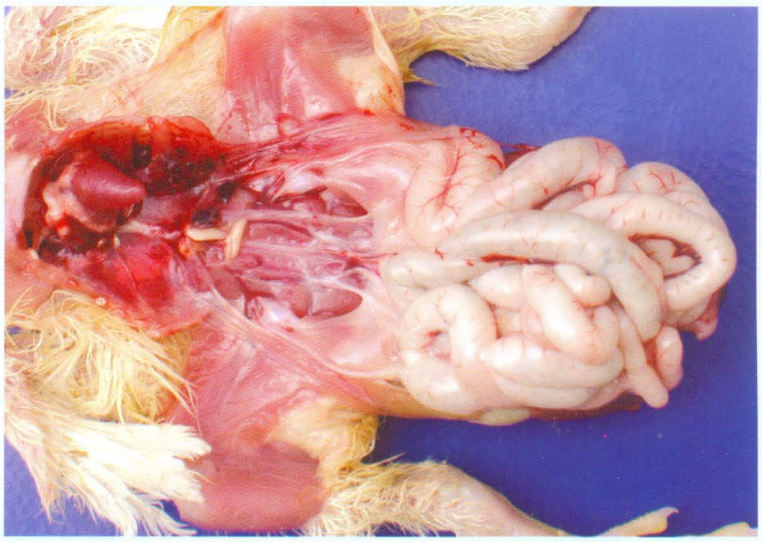
274. Stunting syndrome in broilers. The stunting syndrome in broilers is associated with a reovirus infection but according to some studies, the role of the reovirus is probably secondary. It is characterized by a considerably reduced live weight in affected birds and a various degree of ununiformity in the flock varying from 5-10% to 40-50%, usually seen after the age of 14 days.

275. The growing primary wing feathers are abnormally big for chickens with retarded growth, they protrude at various angles, so the disease is termed **„helicopter disease"**. One-day old chickens are the most susceptible to the infection.



276. Usually, a high-degree atrophy of the pancreas is observed. Reoviruses are shed with faeces and could contaminate the egg shells. The transmission of the infection to susceptible chickens is realized horizontally. The vertical route of transmission is also proved. Reoviruses could persist in infected birds for more than 40 weeks.

277, 278. The small intestine is pale, dilated and contain indigested forage. The tentative diagnosis of viral arthritis and stunting syndrome could be made on the basis of symptoms and lesions. The detection of reovirus antibodies via ELISA supports the diagnosis. Viral arthritis should be differentiated from *M. synoviae*-induced, staphylococcal arthrites and the spontaneous rupture of the tendon of the gastrocnemius muscle. The vaccination of broiler breeder flocks with live or inactivated vaccines protects one-day-old chicks. Infected birds should be removed from the premises. The iodine disinfectants are considered effective for inactivation of agents.



Neoplastic diseases

AVIAN NEOPLASTIC DISEASES

VIRUS-INDUCED NEOPLASTIC DISEASES

A/ DNA herpesvirus neoplastic diseases
Marek's Disease

B/ RNA retrovirus neoplastic diseases:

I. *L/S group of viruses **LSC

1. Neoplasms of the haematopoietic system

Lymphoid leukosis (LL)

Erythroblastosis (ER)

Myeloblastosis (MB)

Myelocytomatosis (MC)

2. Non-haematopoietic neoplasms

Endothelial tumours

Osteopetrosis

Connective tissue tumours

Epithelial tumours

Mixed mesenchymal and epithelial tumours (carcinosarcomas)

II. Group of reticuloendotheliosis viruses (REV)

1. Acute reticular cell neoplasms

2. Runting Disease Syndrome

3. Chronic neoplasms

III. Virus of the lymphoproliferative disease of turkeys (LPD)

NEOPLASMS WITH UNKNOWN AETIOLOGY

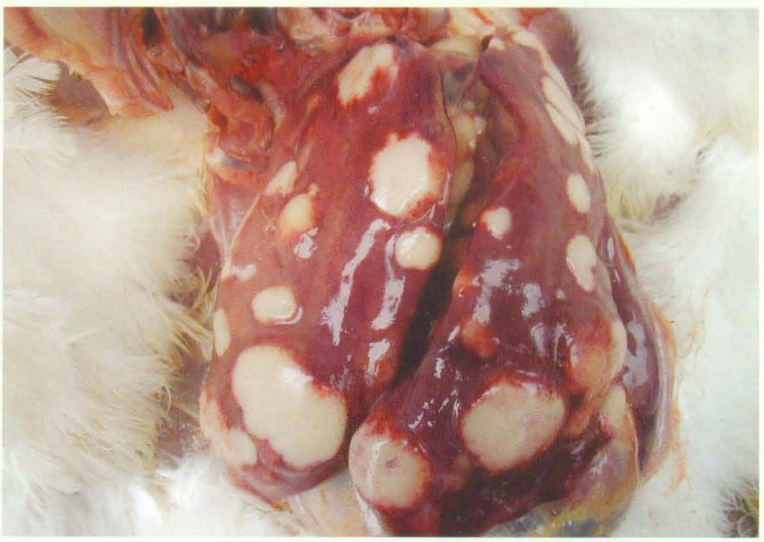
(determined only on the basis of morphological features)

*** Leukosis/sarcoma group**

**** Leukosis/sarcoma complex-associated neoplastic diseases**

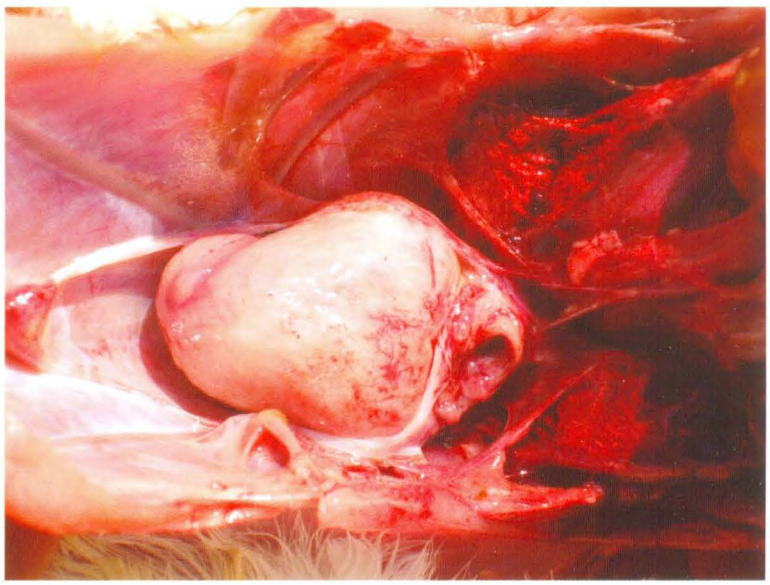
VIRUS-INDUCED NEOPLASTIC DISEASES

MAREK'S DISEASE

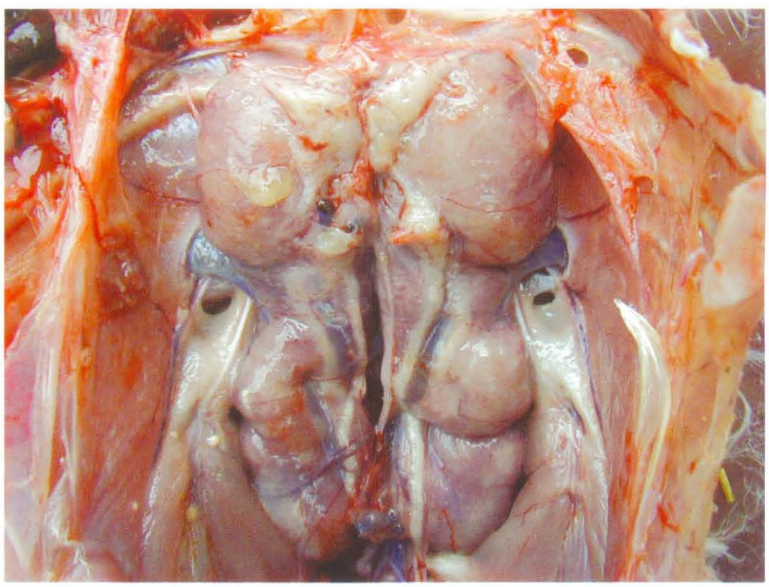


279, 280, 281.
Acute (visceral) form.
It is characterized by diffuse or nodular lymphomatous lesions in various viscera (liver, spleen, heart, kidneys, lungs, gonads, proventriculus, pancreas etc.), the skeletal muscles and the skin. MD affects mainly hens, and is rarely observed in turkeys. It is most commonly encountered in birds at the age of 89 weeks and in layer hens. The cases at the age of 16-20 and 24-30 weeks are predominant. MD is prevalent all around the world and in fact, all flocks are exposed to the effect of the aetiological factor.

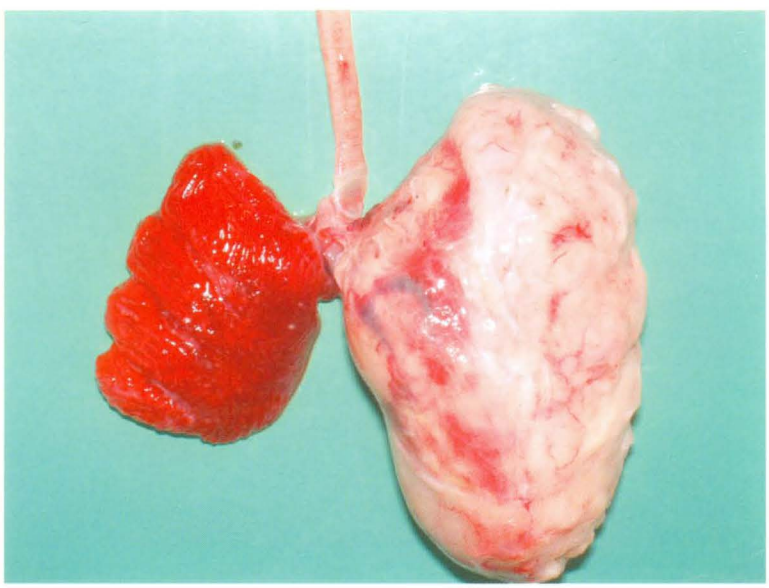
282. Diffuse lymphomatous growths in the heart, resulting in its transformation into an amorphous tumourous mass.

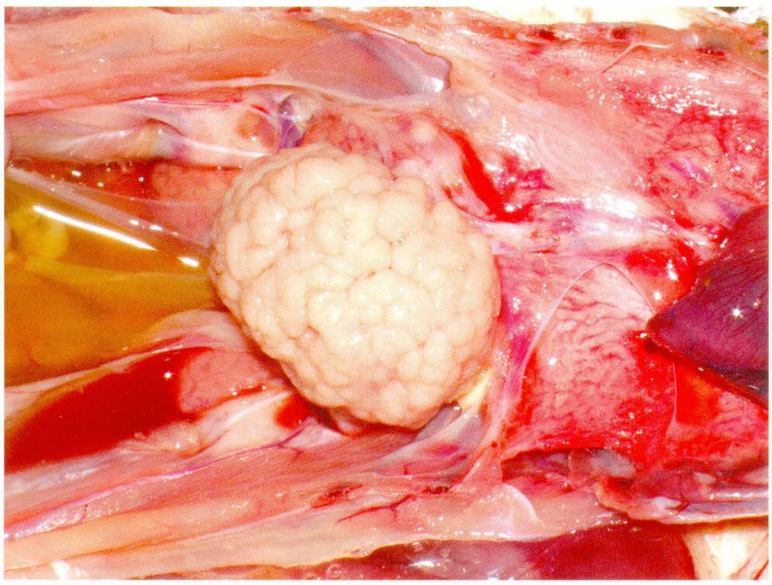


283. Bilateral enlargement of kidneys because of a diffuse lymphoid cell proliferation.



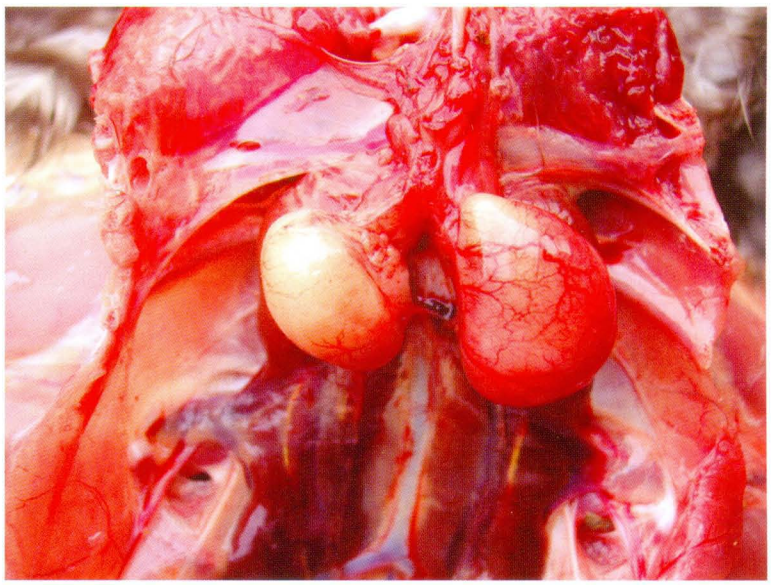
284. Neoplastically modified right lung in MD.



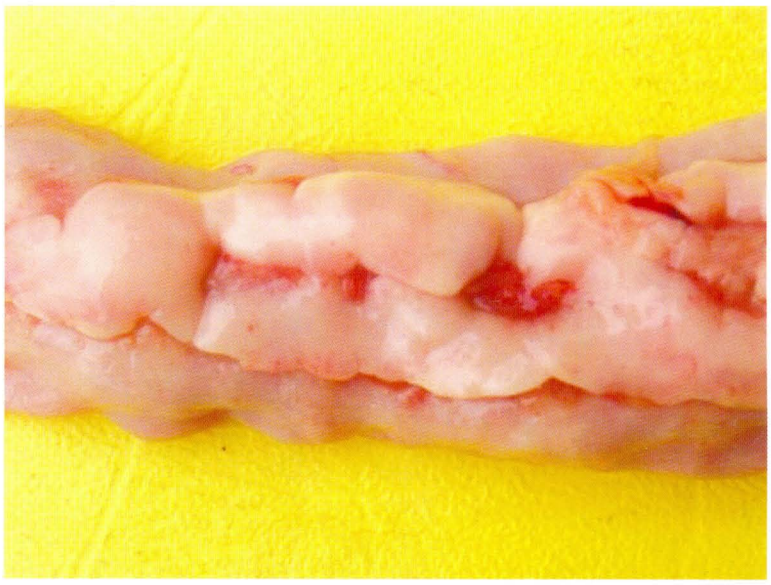


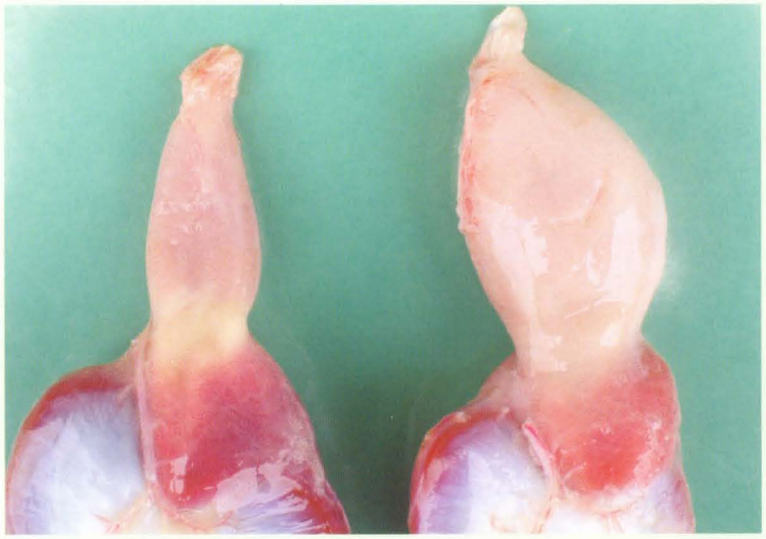
285. Typical cauliflower-like appearance of the ovary, distinctive for MD.

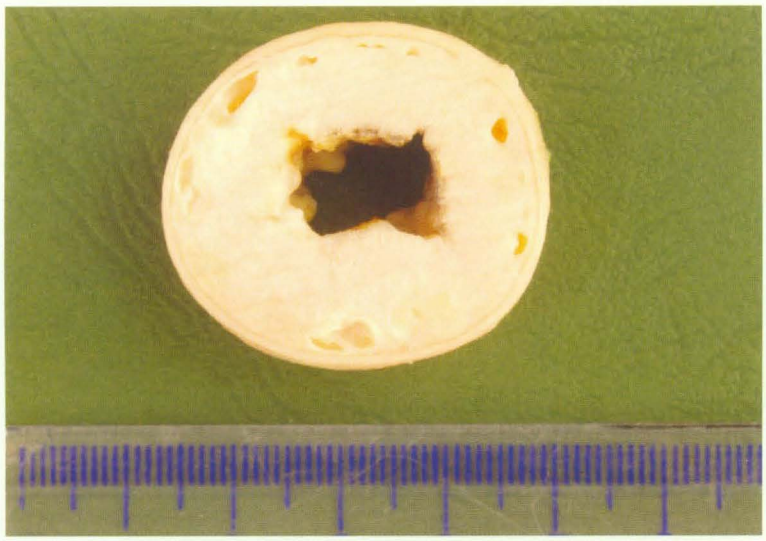
286. Marked asymmetry of testes in a cock following unilateral lymphoid cell proliferation.



287. Diffuse neoplastic growths affecting the pancreas.



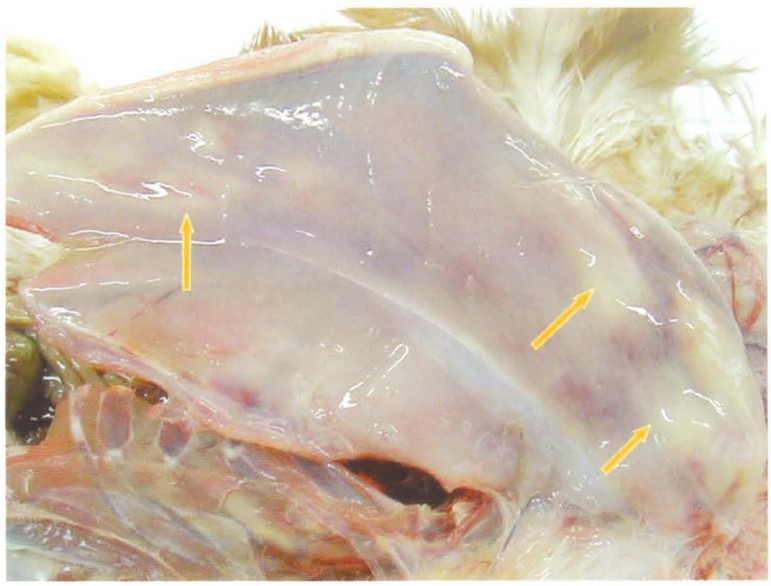




288, 289. The manifold enlarged proventriculus with the shape of a round bottom flask (288) result of diffuse neoplastic growth and the severely narrowed lumen (289) are a typical finding in MD. The causative agent of MD is a type B cell associated herpesvirus (MDV).

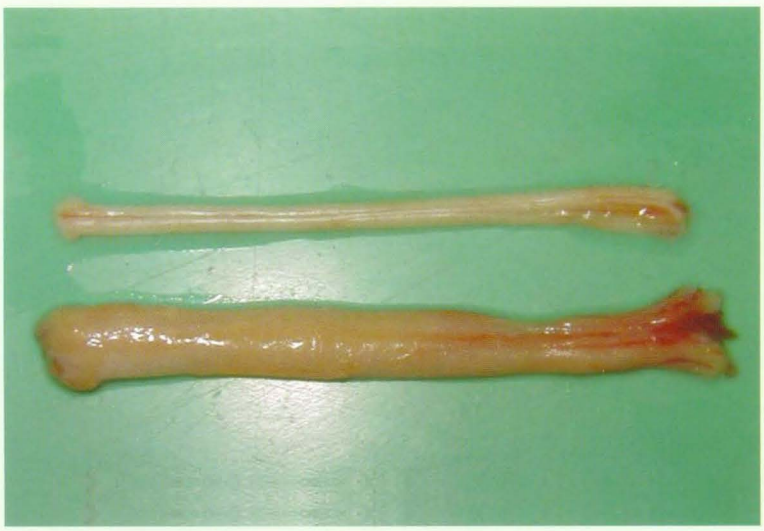
There are three MDV serotypes. The isolates of serotype 1 are widely distributed among hens and vary from highly virulent (vv+) oncogenic to almost avirulent strains. The serotype 2 is common for hens and is not oncogenic. The isolates of serotype 3, known also as turkey herpesviruses (HVT) are naturally occurring in turkeys and are non-oncogenic. The three serotypes possess a significant cross reactivity.

290. Multicentric MD tumours (arrows) prominating or seen through the superficial and deep pectoral muscles.



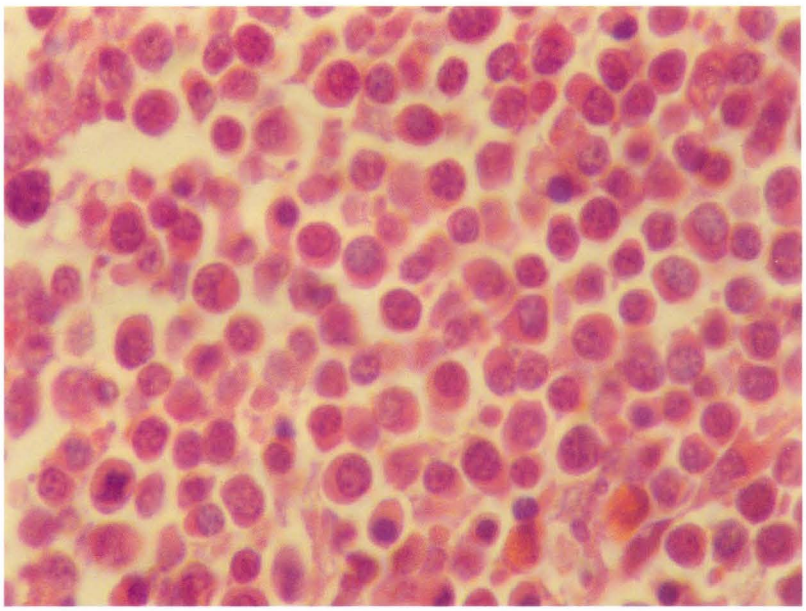
291, 292.
Chronic (classical) form. It is encountered as **neural type (fowl paralysis)** or **ocular type (ocular lymphomatosis)**. Clinically, the neural form is manifested with paralysis of limbs.

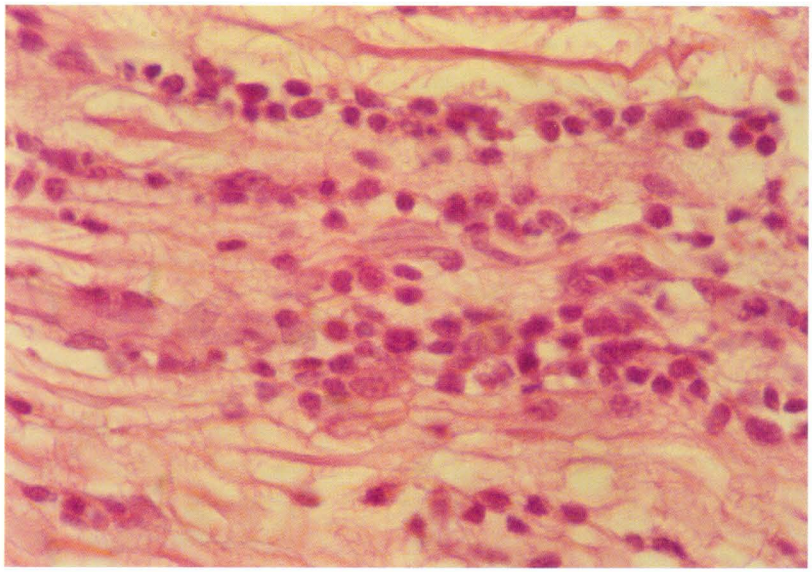
293, 294. Pathoanatomically, unilateral or bilateral thickening of affected nerves, mainly diffuse and at a various extent, is observed.



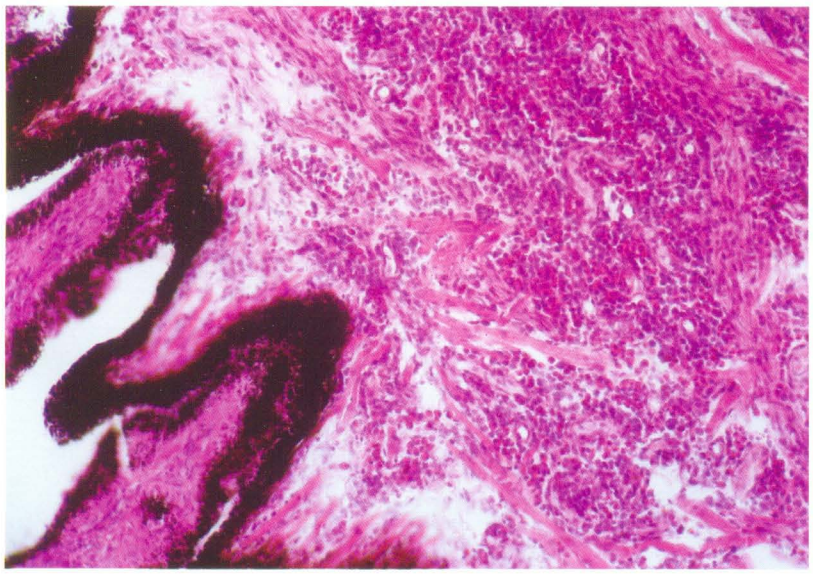
295. The ocular form is characterized with iris depigmentation, deformation of the pupil, sometimes opacity of the cornea and blindness.

296. Histologically, pleiomorphic lymphoid cell proliferations in affected viscera, nerves or eyes are observed. The Marek's disease is probable provided that at least one of the next conditions is present: peripheral nerves augmentation, depigmentation of the iris or irregularly-shaped pupil; lymphoid tumours in various organs in birds younger than 16 weeks; presence of visceral tumours in birds at the age of 16 weeks and older; simultaneous lack of alterations in the bursa of Fabricius.





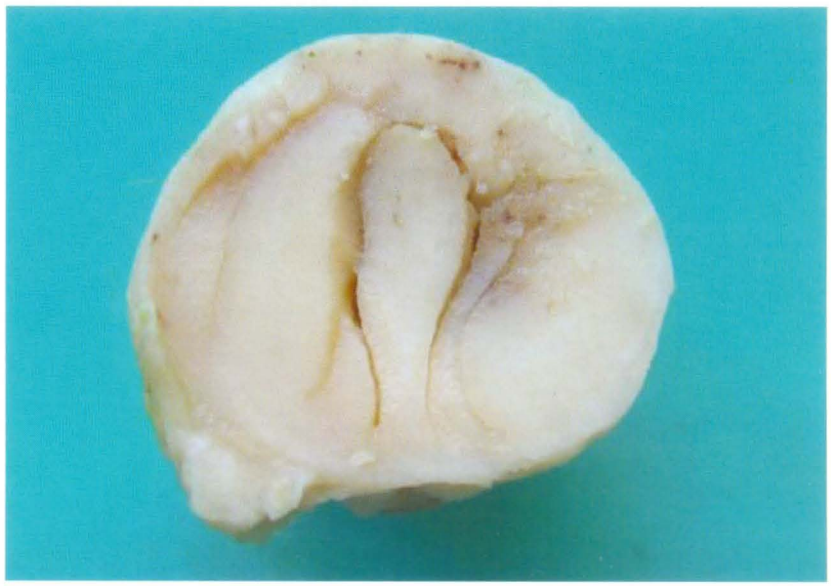
297. Microscopical view of lesions in a peripheral nerve consequent to MD.



298. Lymphoid cell proliferations in the iris and the ciliary muscles in the ocular form of MD. Three classes of viruses are able to protect fowl from MD: attenuated serotype 1 of MDV-cell associated vaccines; HVT could be used for preparation of cell-free lyophilized vaccines; naturally apathogenic isolates of serotype 2 cell associated vaccines. The vaccines against MD provide over 90% protection. HVT gives excellent results but in case of failure, a bivalent vaccine could be used.

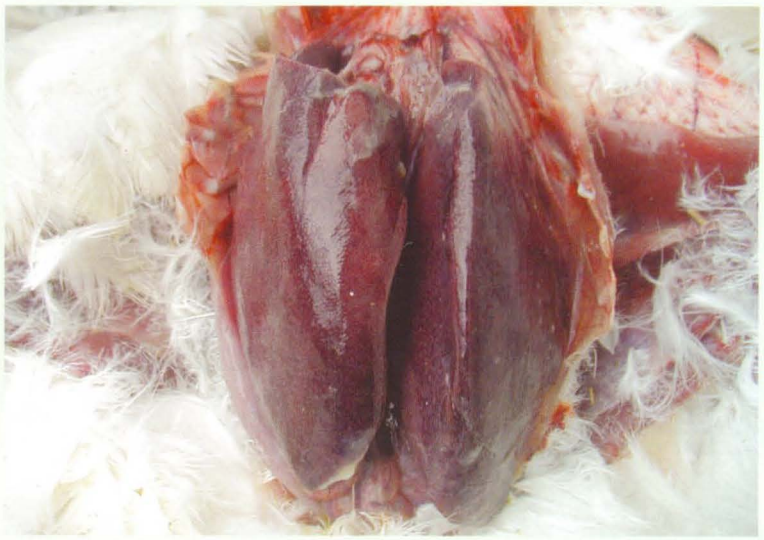
299. Transient paralysis.
They are observed in chickens and hens, especially non-vaccinated against MD. Most cases present the classical form manifested by flaccid paralysis of the neck and legs for 143 days followed by complete recovery. The syndrome has to be differentiated from the neural form of MD on the basis of its transient nature and the flaccid, but not spastic paralysis.

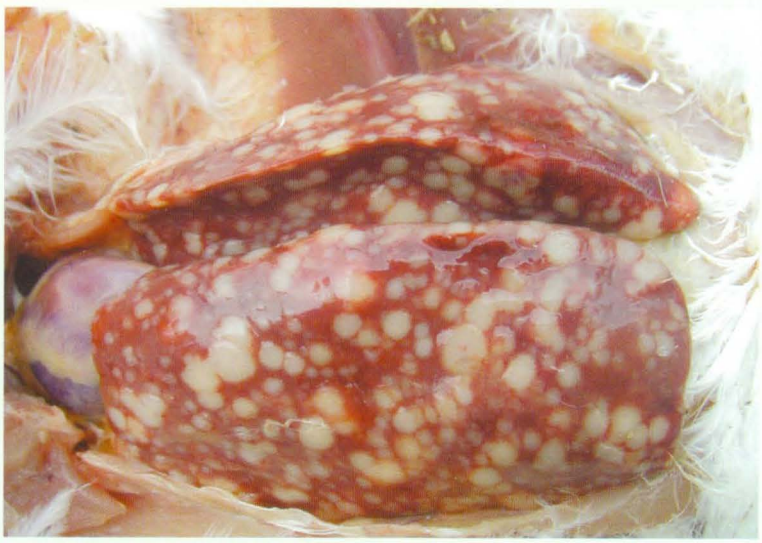
LYMPHOID LEUKOSIS

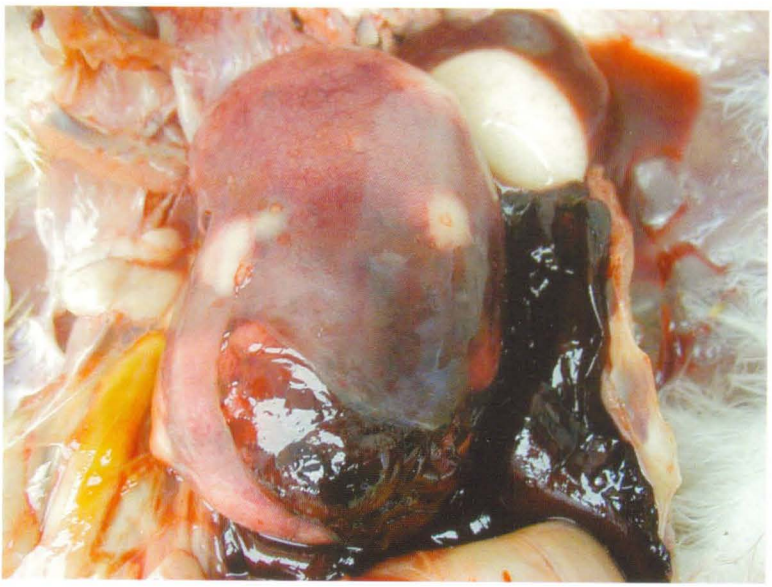


300. It is characterized by a gradual beginning, persistent low mortality in the flock and diffuse or focal neoplastic growths of lymphoblasts in viscera. The neoplastic changes begin always from the bursa of Fabricius, where various-sized lymphomas are detected (transverse section through neoplastically grown bursa - fixed preparation).

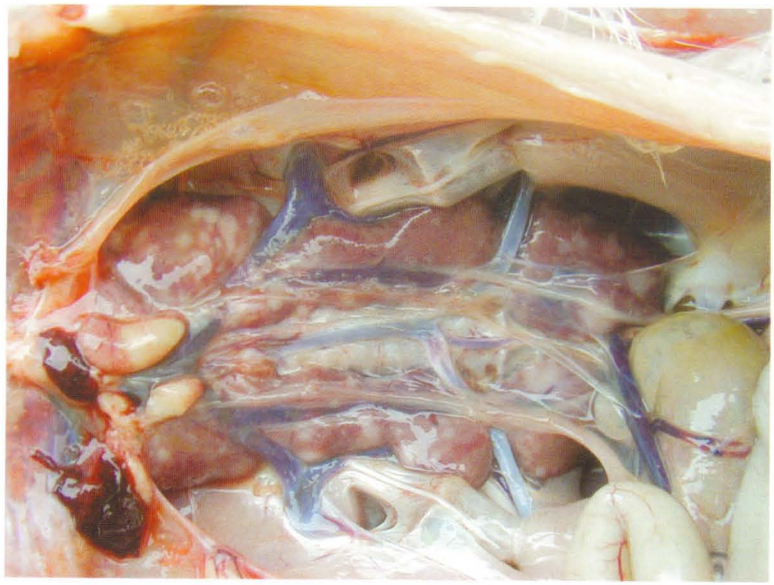
301, 302. Clinically, pale comb and wattles, sometimes swelling of the abdomen because of the highly enlarged liver are observed. Diffuse or nodular neoplastic growths could be detected in many organs, but they are more common in the liver, the spleen, the kidneys, the heart and the ovary.





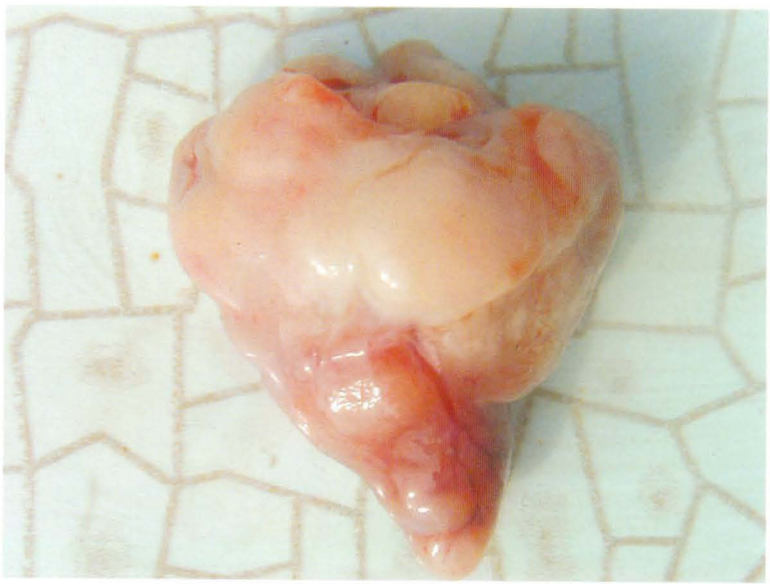


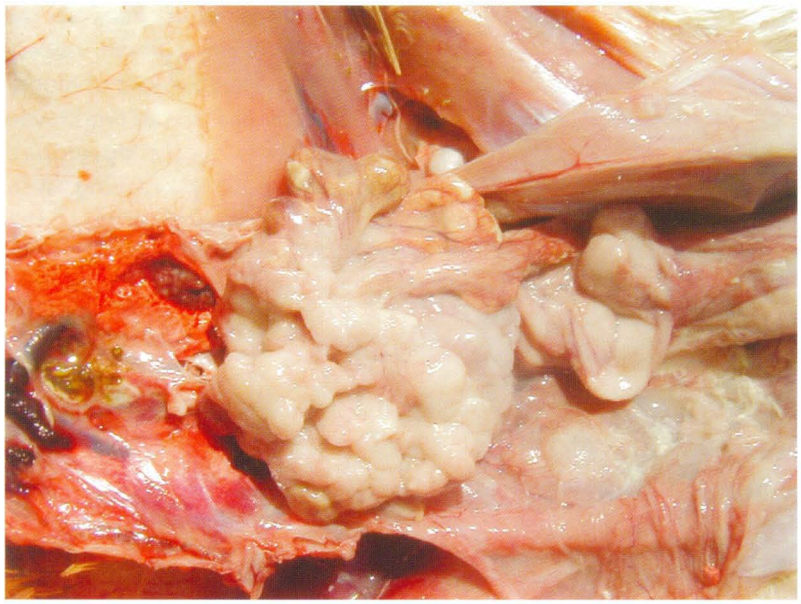
303. Spontaneous rupture of the neoplastically grown spleen, leading to extensive loss of blood. LL is widely distributed worldwide in countries with developed industrial poultry breeding. It is usually observed in birds at the age of 16 weeks and older.



304. Focal neoplastic lesions in kidneys. LL is caused by viruses of the L/S group classified in 10 subgroups: A, B, C, D, E, F, G, H, I and J. The viruses from subgroup A are most prevalent and most frequently associated with LL. Hens, rarely turkeys, pheasants and quails are susceptible.

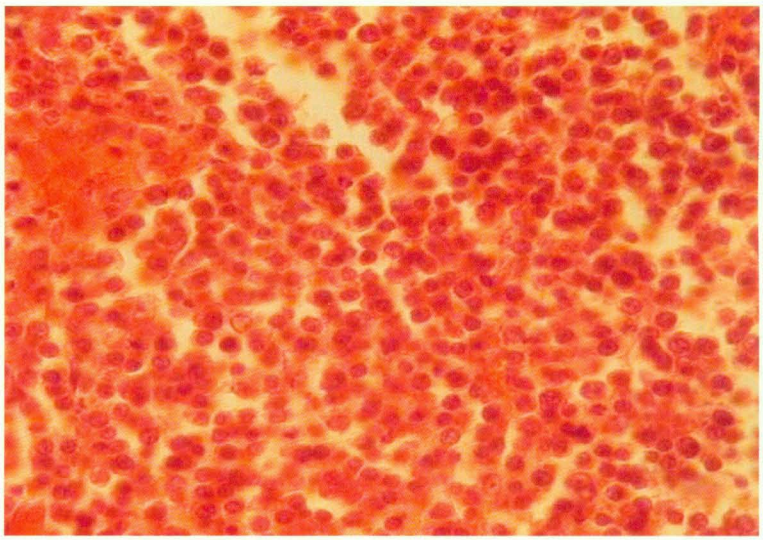
305. Diffuse and focal tumour lesions in the heart. The replication of the virus occurs in albumin secreting glands of the oviduct. The transmission of the infection is performed vertically by egg albumin from one generation to another. The role of cocks is not important for the congenital infection of the progeny. They are only virus carriers and source of venereal infection for other birds.

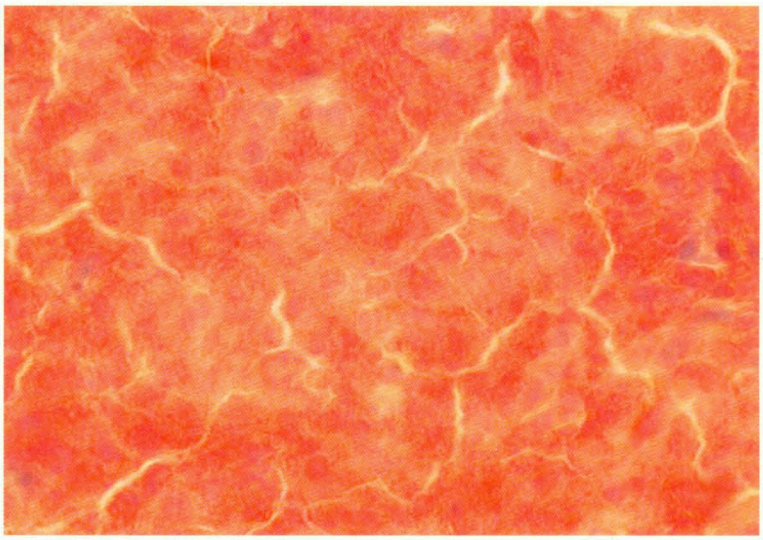


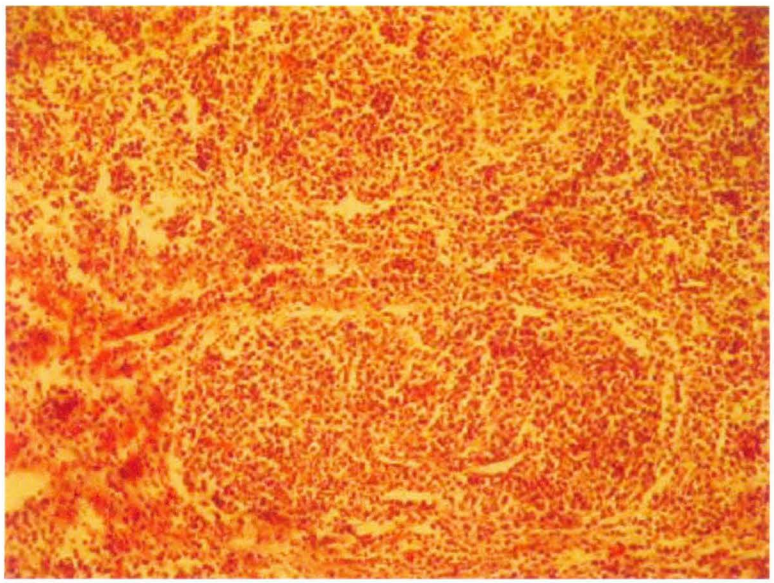


306. Neoplastically transformed ovary in LL. In some instances, the horizontal infection is also possible but only in chickens in the first few days after hatching, usually via vaccines contaminated with ALSV. The lethal issues are observed for 56 months after the LL outbreak and amount to 5 - 15%.

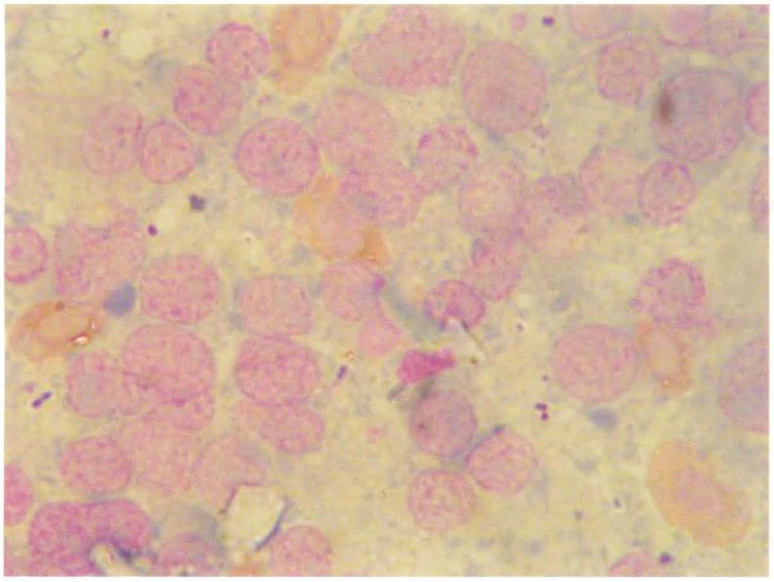
307, 308. Histologically, growth of single type lymphoblast cells with marked pyroninophilia is observed.







309. In the bursa of Fabricius, a characteristic intrafollicular hyperplasia is observed.



310. The picture of an imprint preparation from neoplastic lesions shows a layer of single-type lymphoblast cells. LL and MD are hard to be **distinguished**: in both, lymphoid tumours are present in the same visceral organs, the appearance at the same age is possible, and the visceral lesions could not be differentiated macroscopically, except in a careful microscopic examination by an experienced pathologist.

FROM THE POINT OF VIEW OF DIFFERENTIAL DIAGNOSIS, THE FOLLOWING FEATURES DESERVE A SPECIAL EMPHASIS:

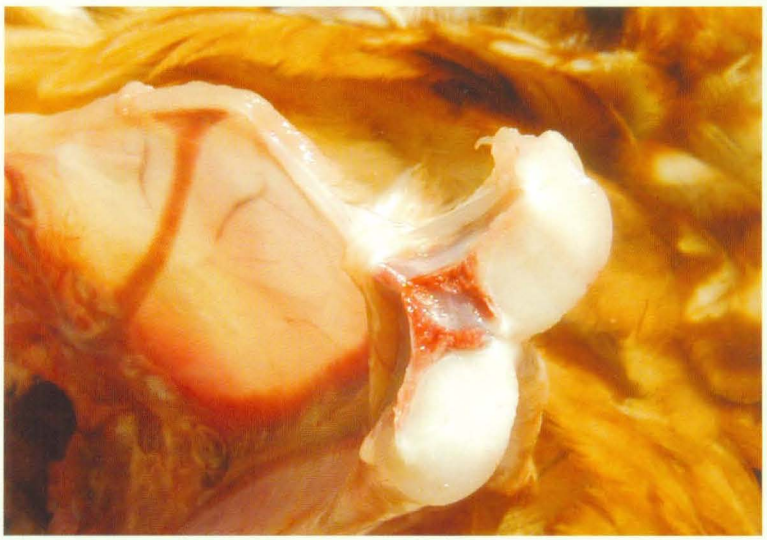
LYMPHOID LEUKOSIS

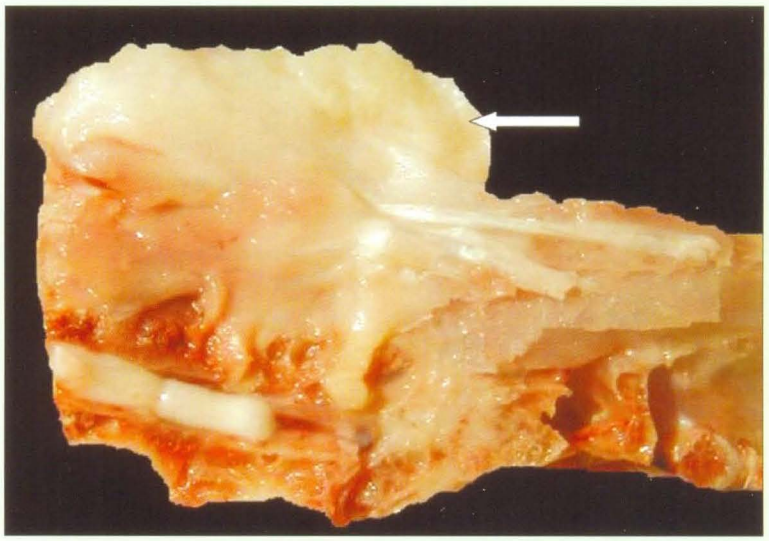
- Usually, it is not seen in birds younger than 14 weeks.
- The lethal issues occur mostly at the age between 24 and 40 weeks.
- Distinct nodular tumours.
- Tumours in the bursa of Fabricius.

MAREK'S DISEASE

- Could be observed after the age of 4 weeks too.
- The peak mortality is seen between the 10th and the 20th week, sometimes continues after the 20th week.
- Paralysis.
- „Grey eye".
- In some birds, the bursa of Fabricius is atrophied, in others: neoplastic.

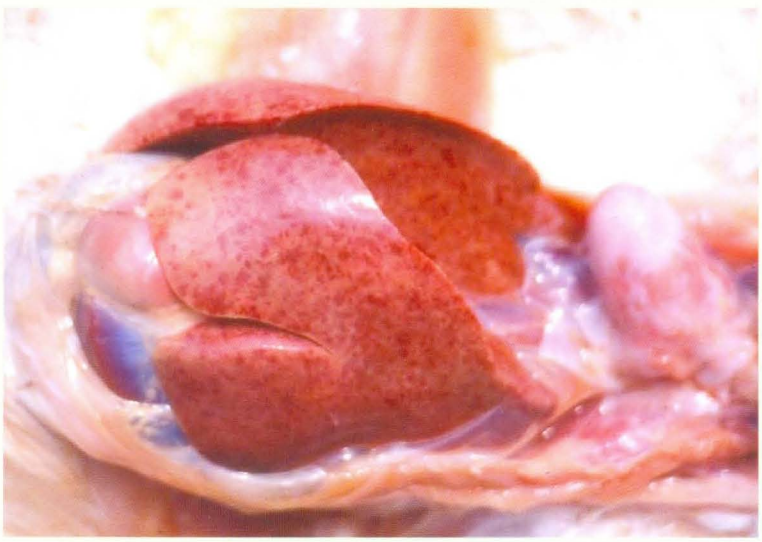
MYELOCYTOMATOSIS

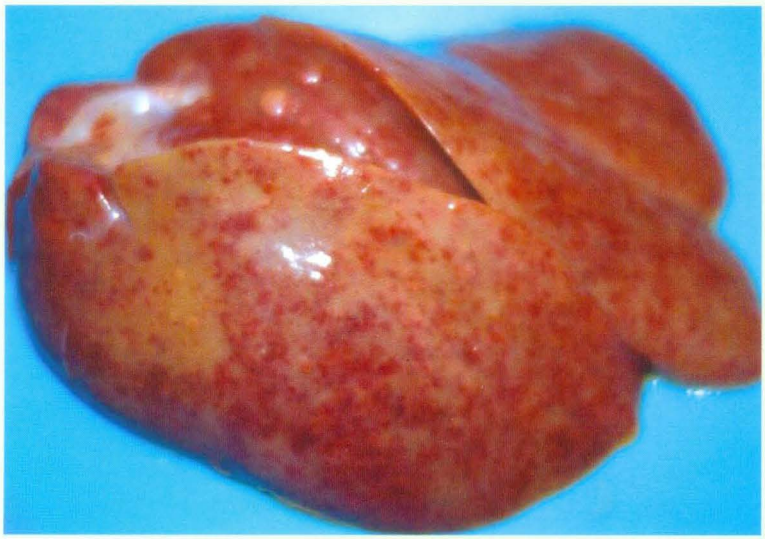




311, 312. Myelocytomatosis (MC) is characterized by proliferation of immature cells from the granulocyte order myelocytes and pro-myelocytes. It has an aleukaemic character. Occurs independently or in association with a number of other neoplastic diseases. Atypical morphological forms are possible. The MC tumours (myelocytomas) are frequently encountered on the bone surface near the periosteum, the adjacent cartilage or bone-cartilage ends of the ribs.

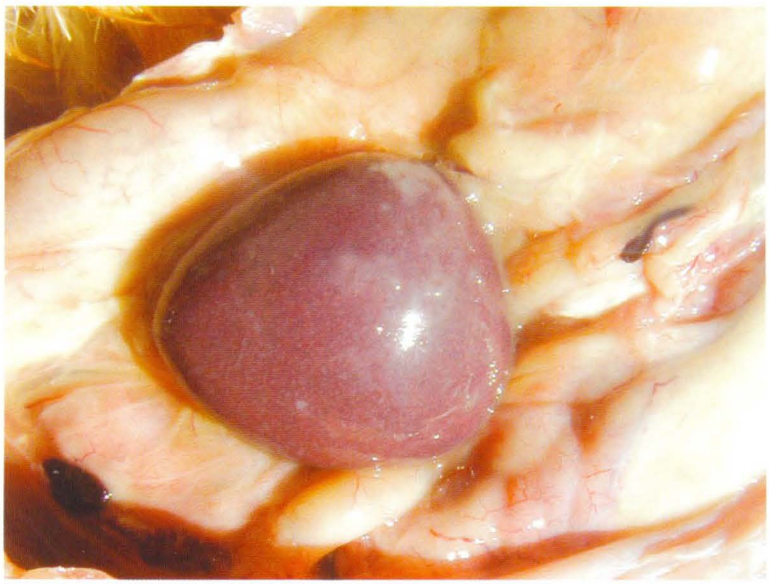
313, 314. MC is caused by viral strains of ALSVs from subgroups A, B and J (MC29, MC31, CMII, OK10, HRPS 103, and ADOL HC1). It is encountered relatively infrequently. Its occurrence is sporadic or enzootic. Susceptible birds are hens, pheasants, guinea hens and quails. In most cases, the liver is enlarged, thick and mottled with dark red spots or fat-like nodules.





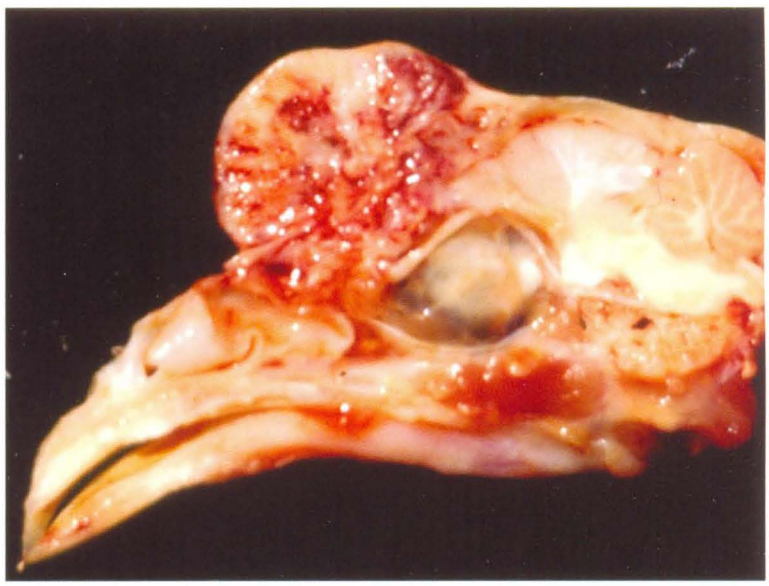
315. Sclerotic changes in the liver are possible because of regression of neoplastic lesions.

316. The spleen is usually enlarged, but sometimes, could be atrophied.

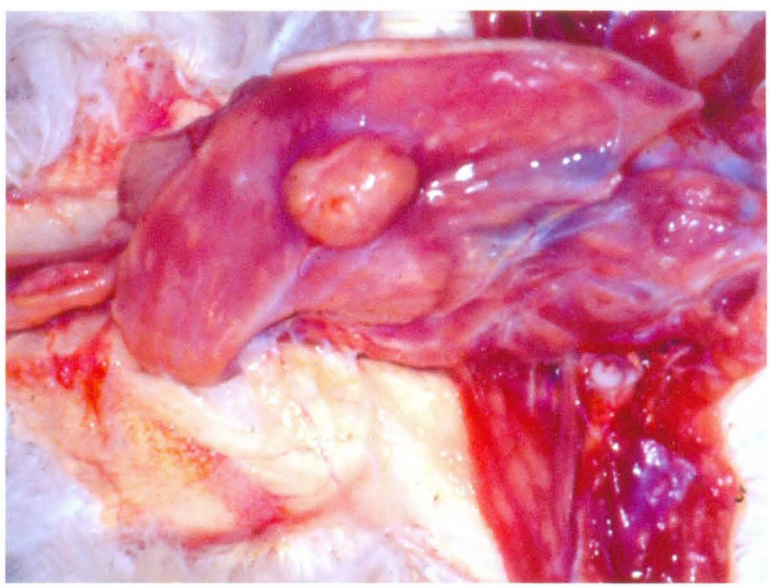


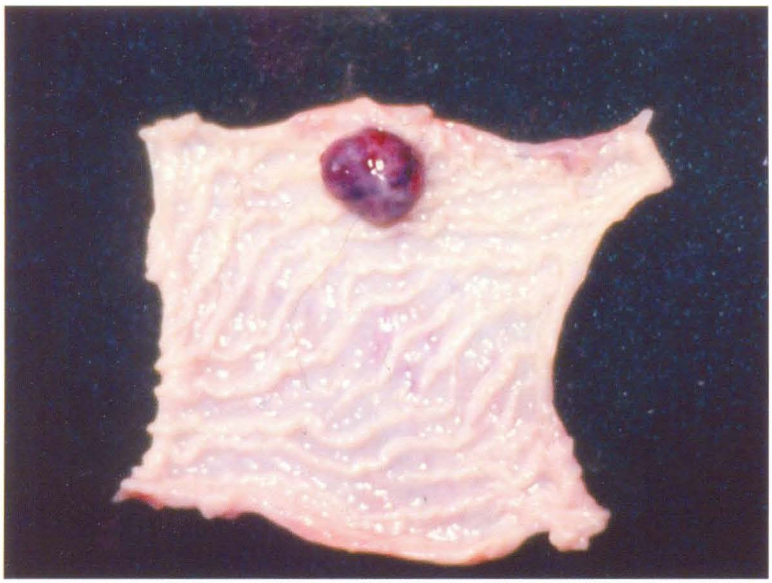
317. A characteristic feature of MC is its simultaneous course with tumours from a different type: mesenchymal, epithelial or mixed. The picture shows a fibrosarcoma to the gizzard associated with MC.

318. Mixed mesenchymal tumour (osteochondrosarcoma) to the frontal skull bones: a sagittal cross section.

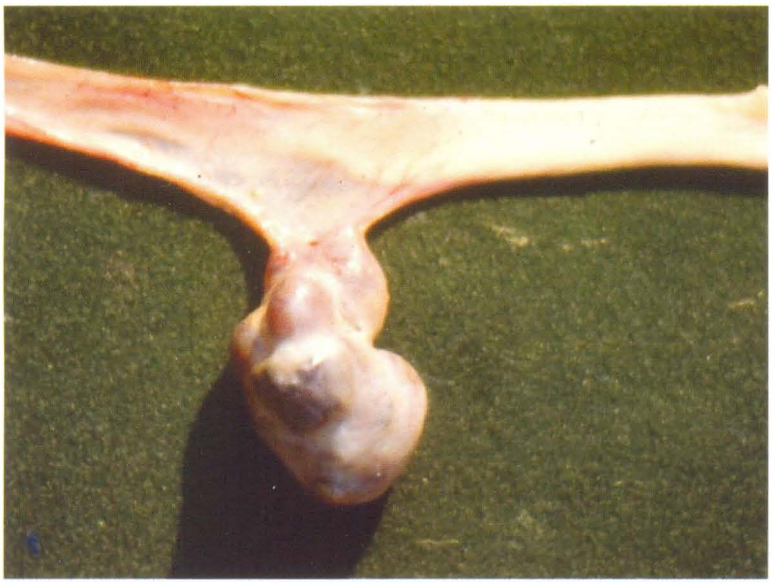


319. Multiple rabdomyosarcoma in pectoral, thigh, abdominal and tracheal muscles.



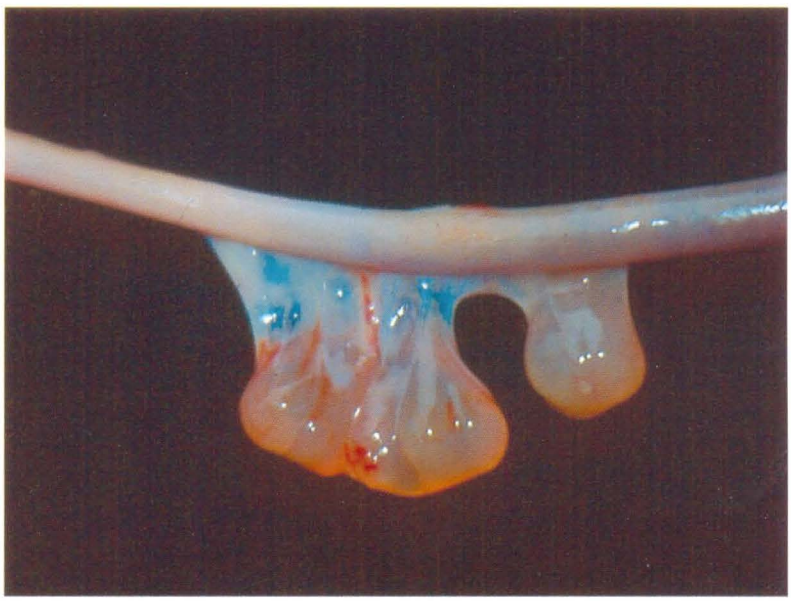


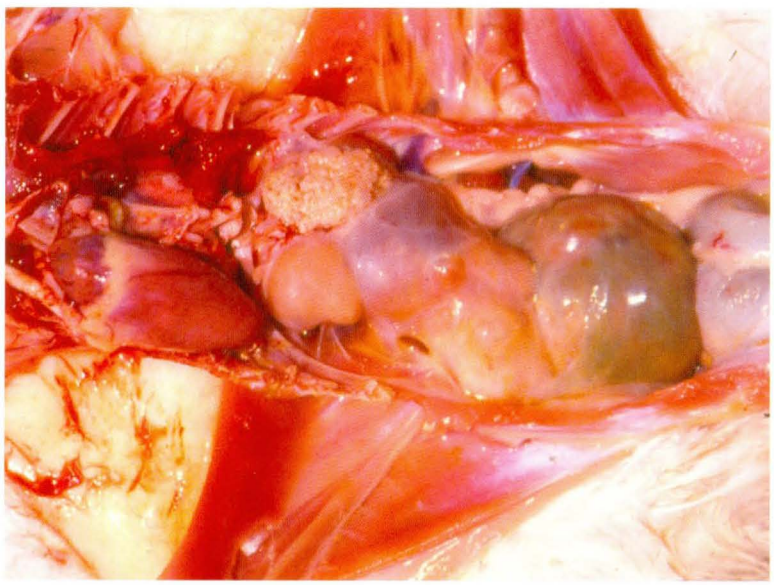
320. Leiomyosarcoma of the mucous coat of the oviduct.



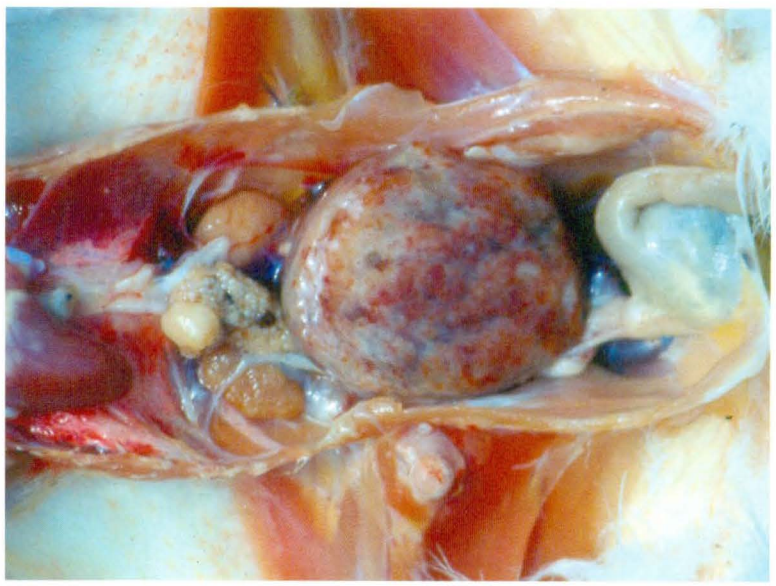
321. Pendulating haemangiosarcoma of the ileal serosa.

322. Pendulating multiple myxoma of the small intestine's serous coat.

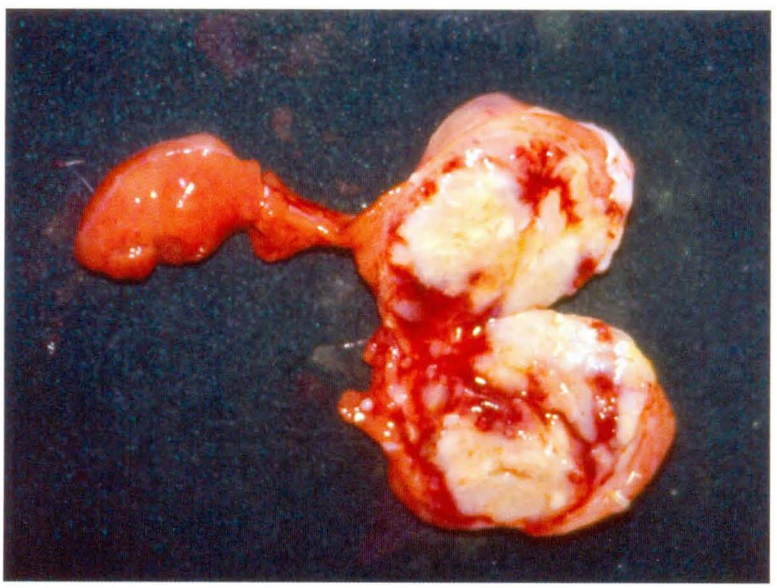




323. MC-associated cystadenocarcinoma of the kidney in a hen.

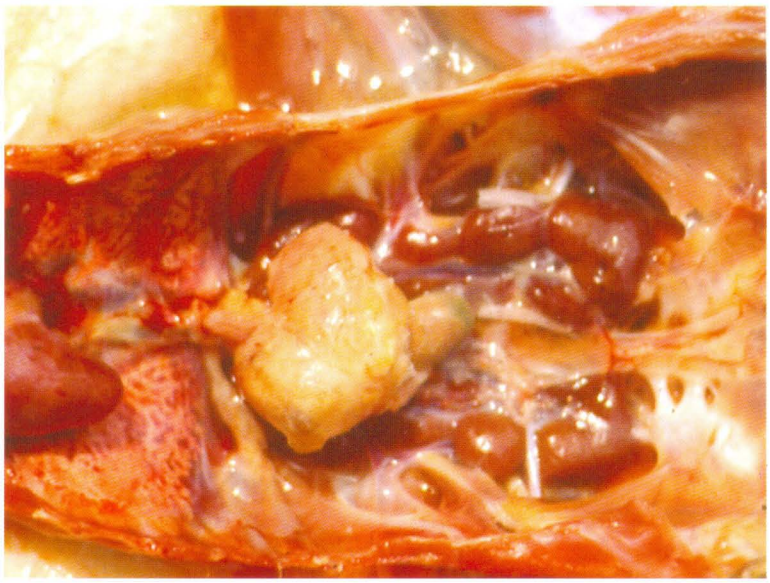


324. Nephroblastoma of the left kidney, occupying a significant part of the abdominal cavity.

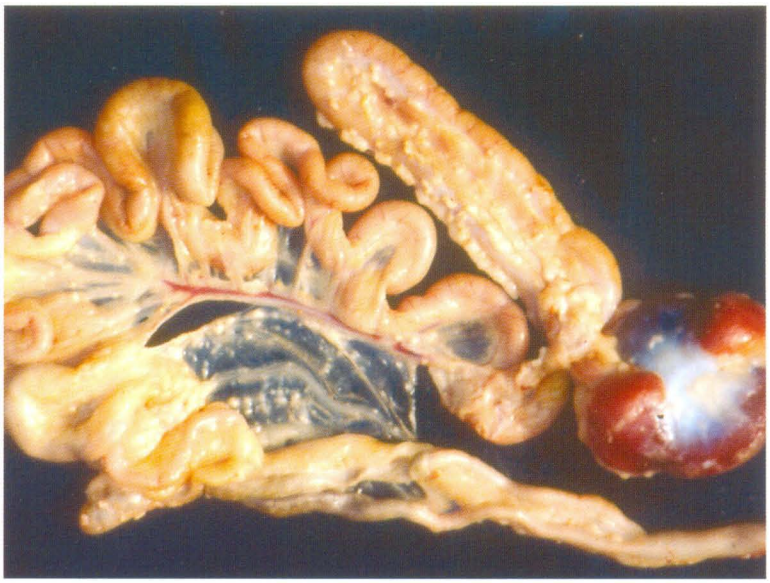


325. Nephroblastoma - the surface of a cross section. The tumour is a pendulating mass attached to the kidney by a fibrous vascularized stem that has undergone a partial necrosis and haemorrhages

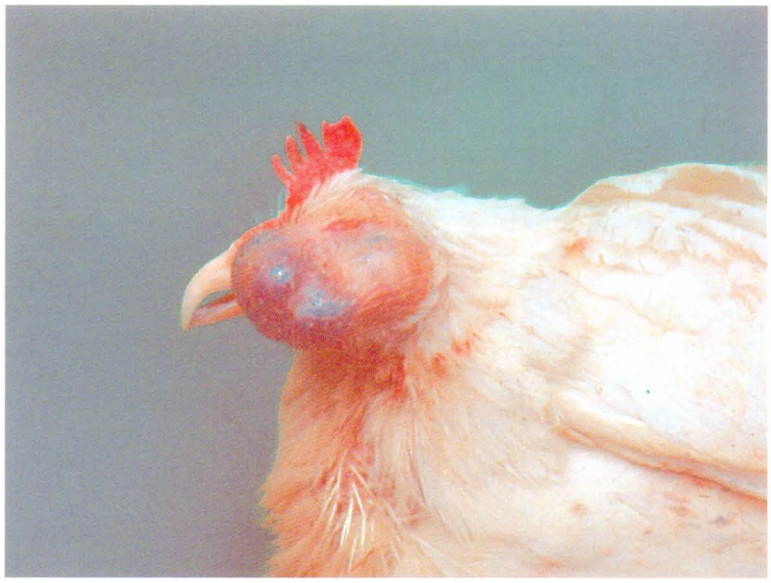
326. Granulosa cell tumour of the ovary. The tumour appears as a single, compact, dorsoventrally flattened growth.



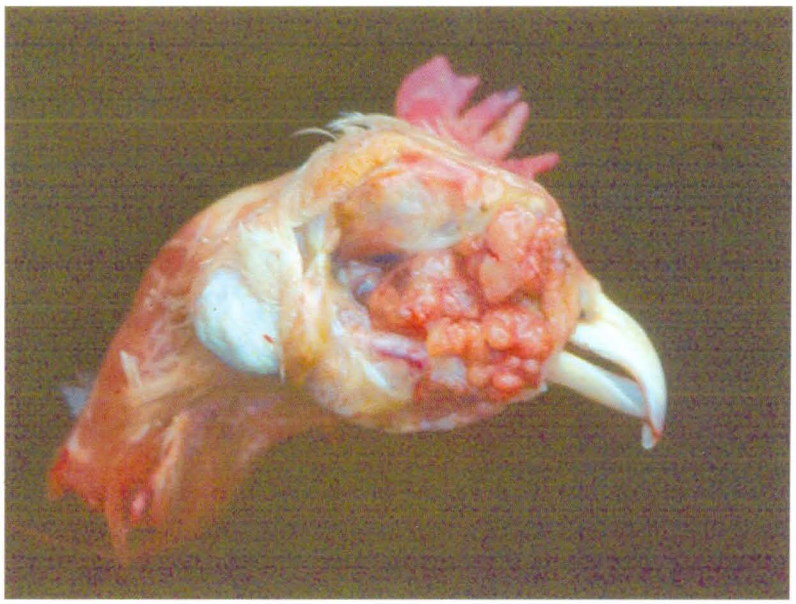
327. MC-associated multiple carcinosarcoma of the mesentery and alimentary tract's serous coat (disseminated milliary nodules).

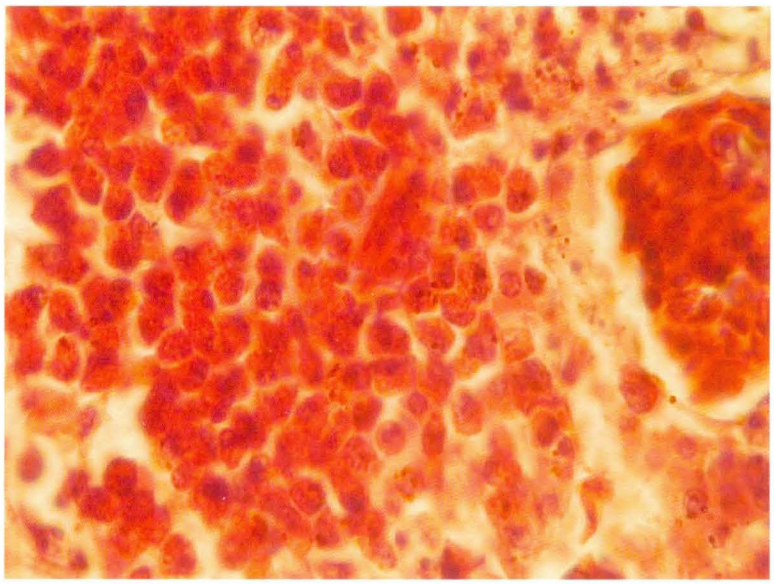


328. MC-associated carcinosarcomas in the region of the right infraorbital sinus.

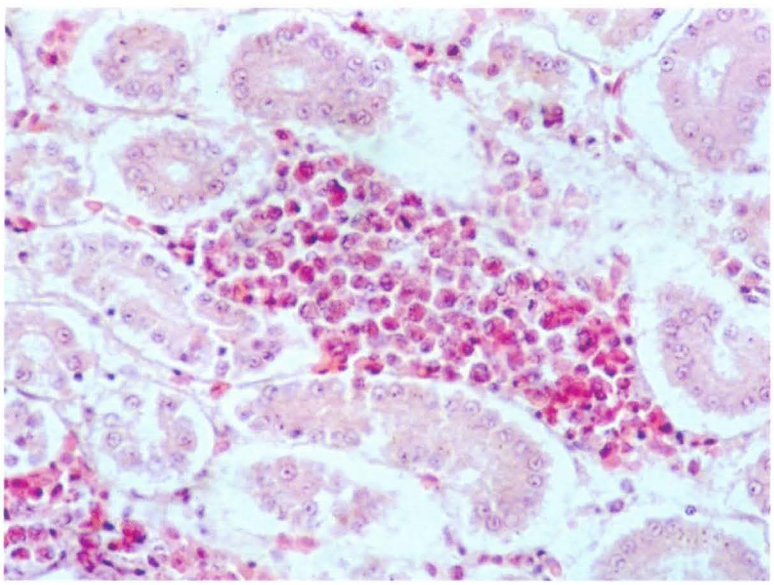


329. Gross appearance of the tumour from Fig. 328 after removal of the covering skin.



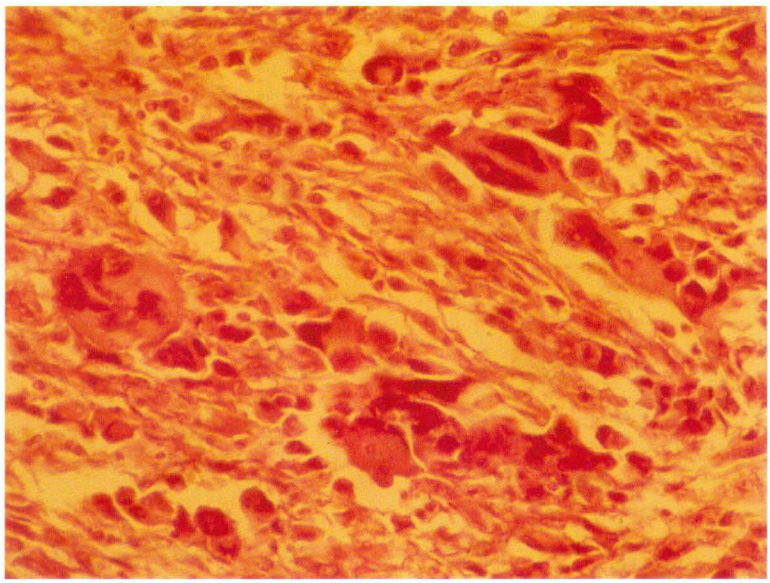


330. Histologically, myelocytomatomas are easily distinguished. Most commonly, they have perivascular localization. Growth of myelocytes with well-formed granules in a liver cross-section.

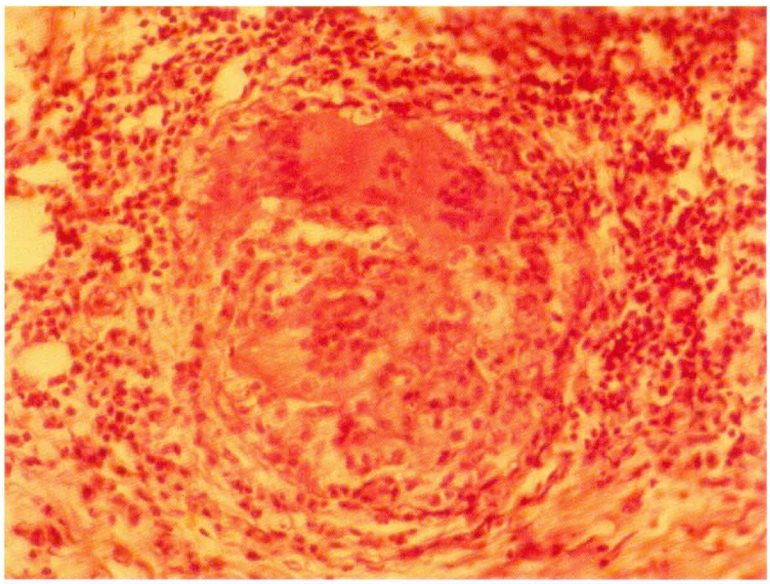


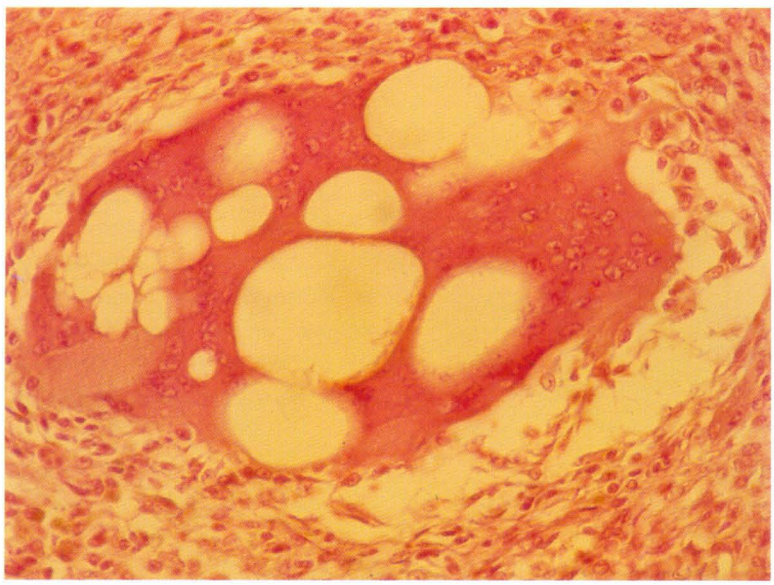
331. Kidney.
Focal intertubular myelocytic proliferations.

332. MC-associated neoplasms of epithelial, mesenchymal or mixed type demonstrate the respective type of histological structure. Leiomyosarcoma a histological view. Polygonal giant cells with hyperchromatic nuclei.



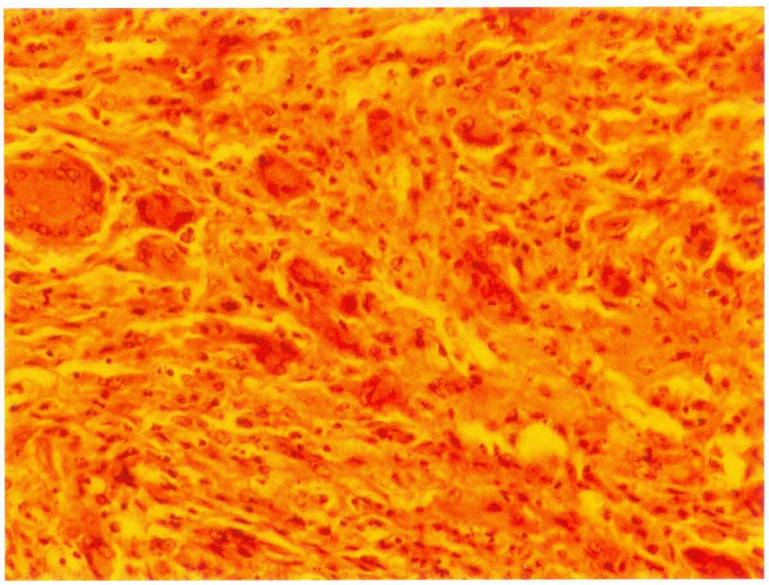
333. Leiomyosarcoma - small intestine. Prolongations of polynuclear symplastic elements.



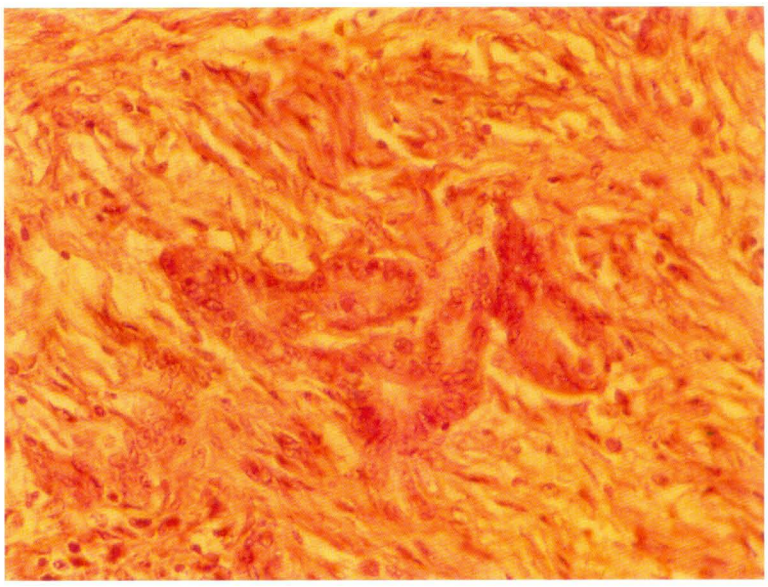


334. Leiomyosarcoma - small intestine. Extraordinary („monstrous") multinuclear giant cell with intracytoplasmic vacuoles.

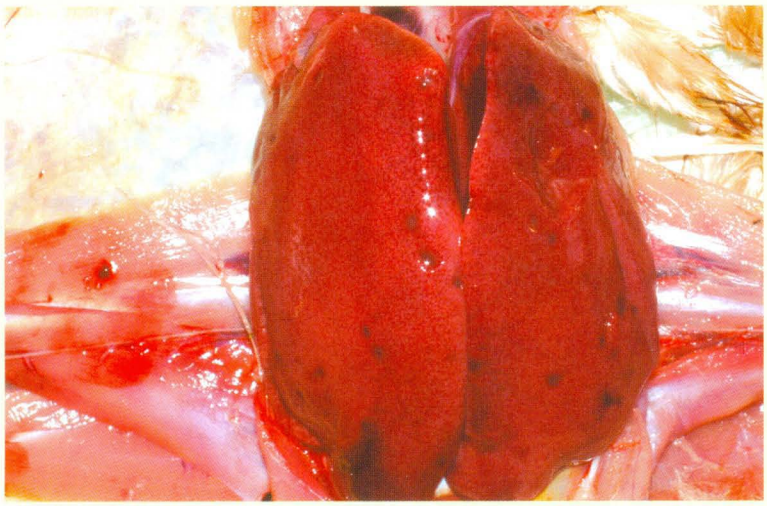
335. Rabdomyosarcoma. An area with multiple hyperchromatic giant cells.



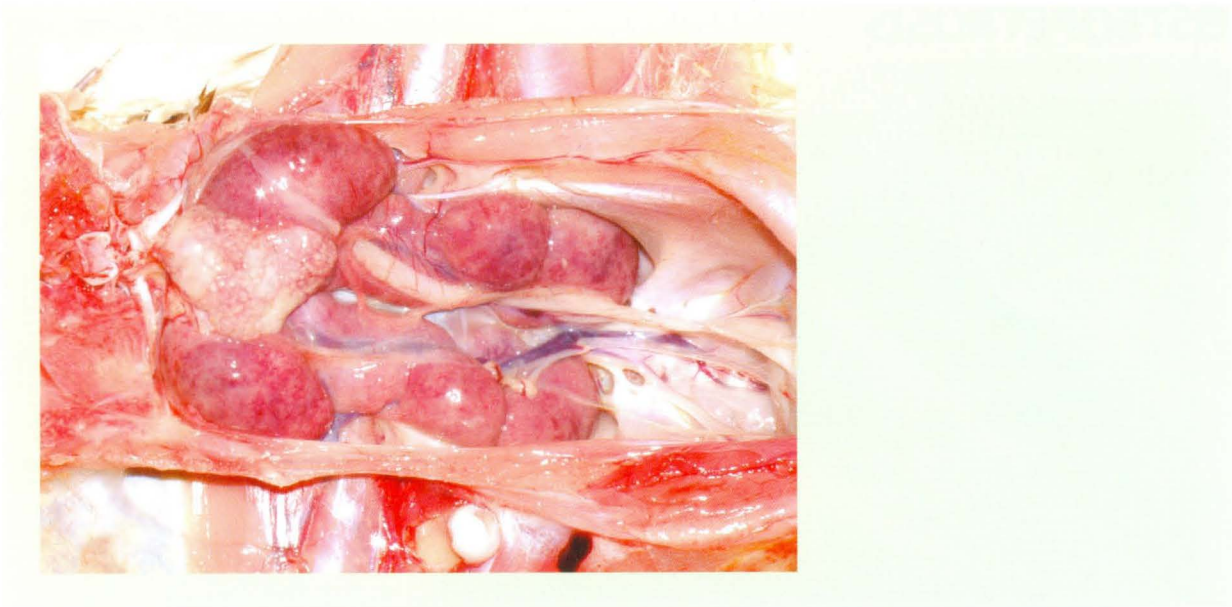
336. Carcinosarcoma of the pancreas. Tubulous glandular epithelial formations of the carcinoma component among the liposarcoma part of the parenchyma. The diagnosis is based upon the entity of data about the history, the gross appearance and location of the tumours and the specific histological lesions. From a differential diagnostic point of view, myeloblastosis and erythroblastosis should be considered.



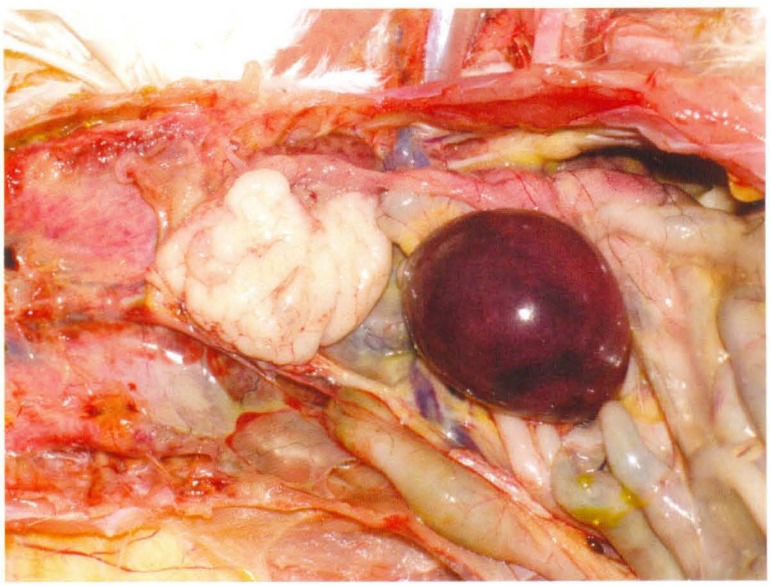
ERYTHROBLASTOSIS



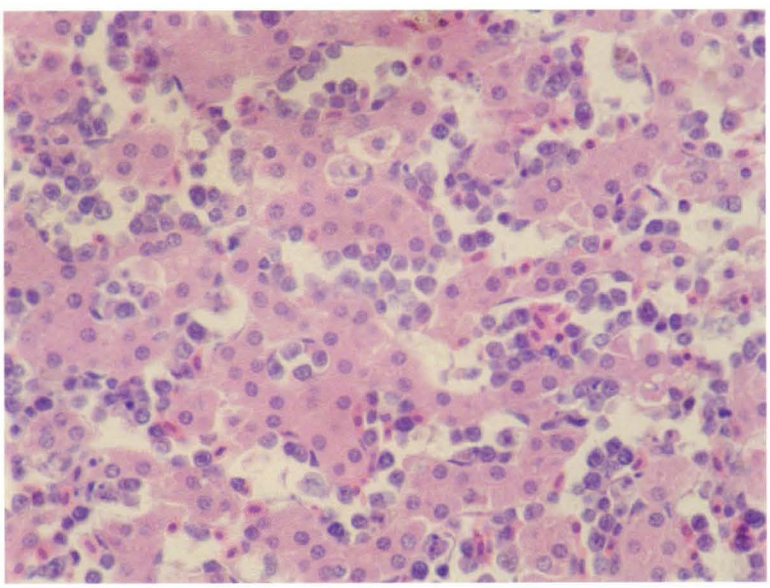
337, 338. Erythroblastosis (ER) is characterized by intravascular proliferations of immature precursors of erythrocytes. ER has a leukaemic character and is manifested with signs of severe anaemia. The liver and the kidneys are moderately enlarged with a characteristic dark red to mahogany colour, sometimes with haemorrhages.



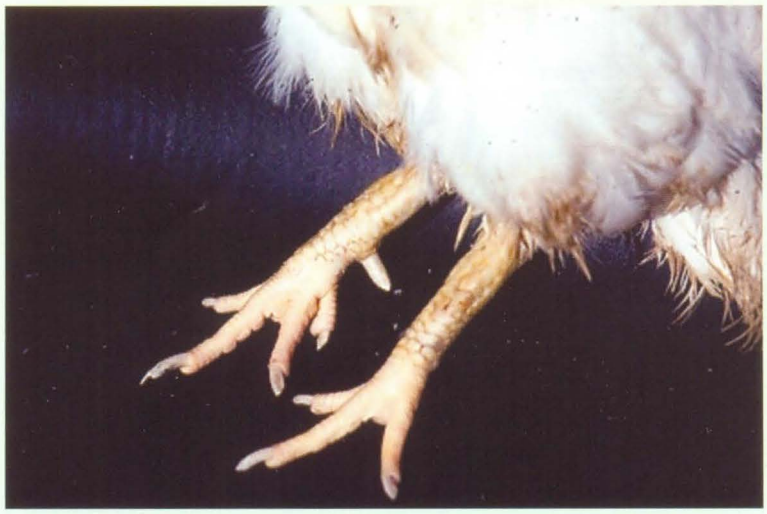
339. ER is caused by the avian eryhtroblastosis virus (AEV); the most frequently encountered strains are E-26, ES4, R etc. The spleen is unusually enlarged or atrophied in cases of severe anaemia.

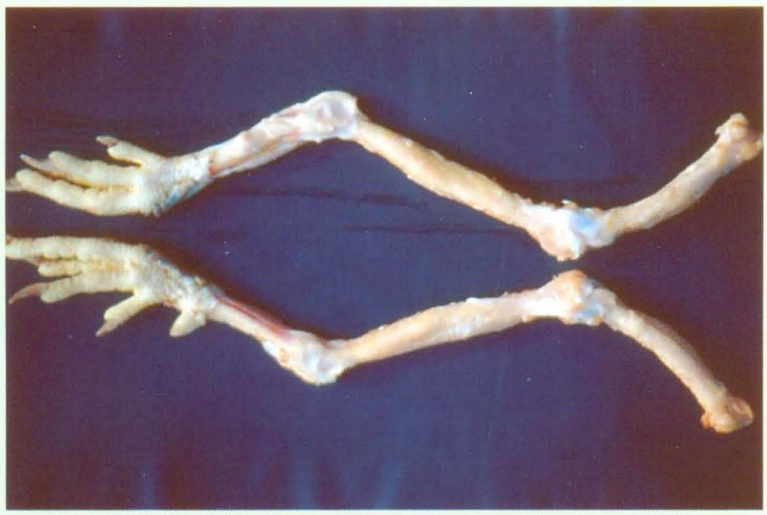


340. Histologically, accumulation of erythroblasts in blood sinusoids and capillaries is seen. The **diagnosis** is based on visceral histological lesions, typical for ER and peripheral blood haematological and morphological analysis.



OSTEOPETROSIS

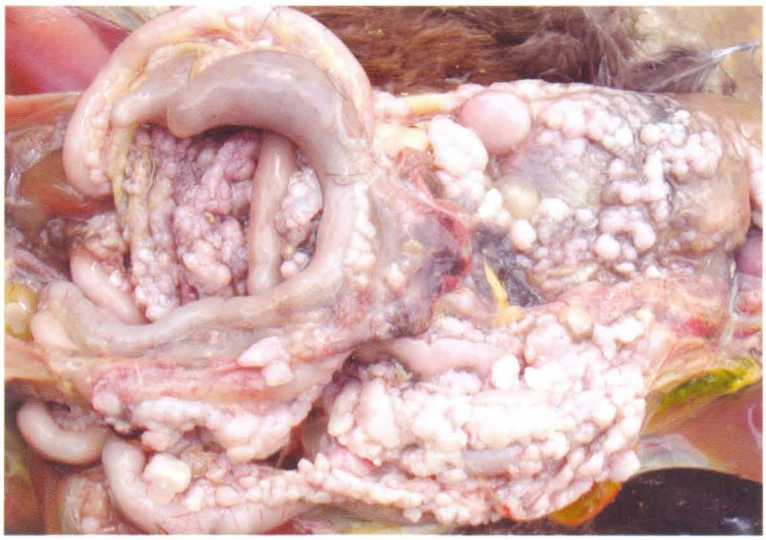




341, 342. Osteopetrosis is a neoplastic disease, aetiologically related to the L/S group of viruses. It is characterized by a significant thickening of bone periosteum. The diaphyses of the tibia and/or tarsometatarsal bones are most commonly affected. Often, osteopetrosis is seen simultaneously with LL in the same bird.

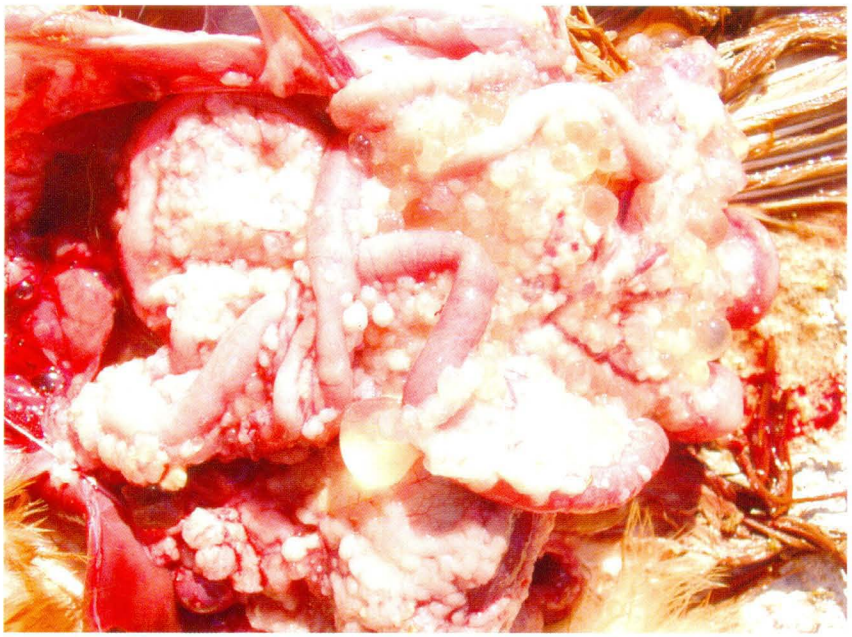
NEOPLASTIC DISEASES WITH UNKNOWN AETIOLOGY

ADENOCARCINOMATOSIS

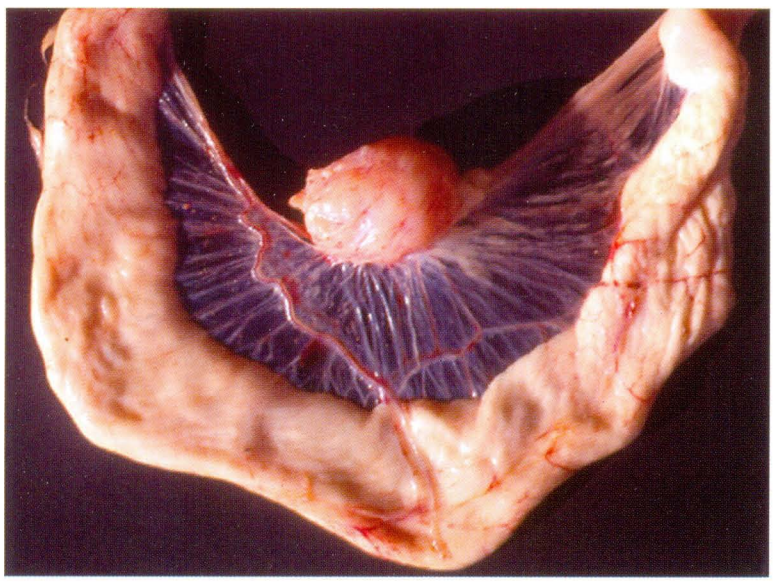


343. The intestinal and reproductive tracts are primarily affected. Quite often, the neoplasms invade the peritoneum and other serous coats. Macroscopically they appear as numerous disseminated thick nodes of a various size (with a diameter from several mm to 1 cm).

344. Sometimes, the tumours are cystic formations (cystadeno-carcinoma).



LEIOMYOMA OF THE MESOSALPINX



345. The leiomyoma of the mesosalpinx is a common tumour in hens. Usually, it is located in the ventral ligament of the oviduct. Its size varies from small to large (several cm in diameter), smooth, thick, sometimes highly vascularized nodes.

Parasitic diseases

COCCIDIOSIS

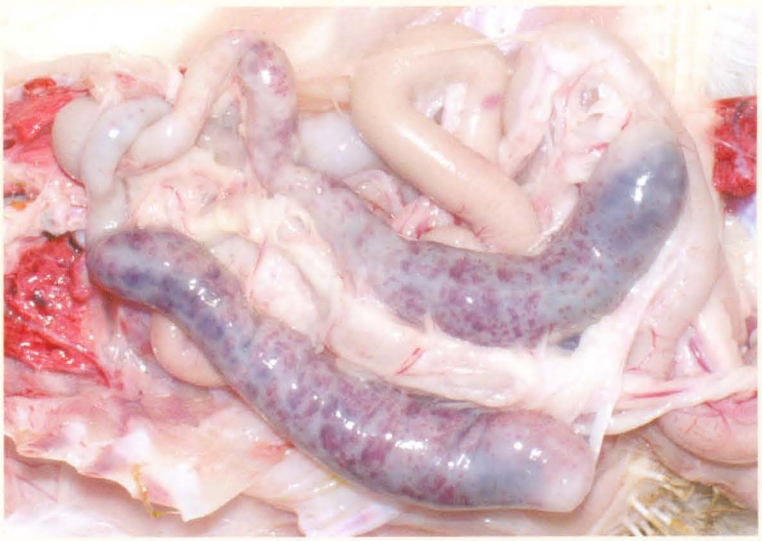
346. Coccidiosis is a common protozoan disease in domestic birds and other fowl, characterized by enteritis and bloody diarrhoea. The intestinal tract is affected, with the exception of the renal coccidiosis in geese. Clinically, bloody faeces, ruffled feathers, anaemia, reduced head size and somnolence are observed.

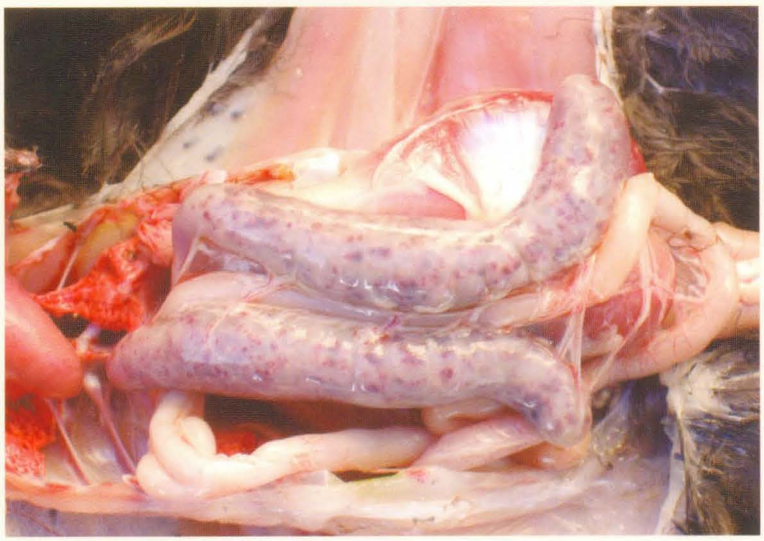
347. The area around the vent is stained with blood. The infection is realized by a faecal-oral route. After ingestion of sporulated (infective) oocysts, sporozoites are released that enter asexual and sexual cycles of development resulting in the emergence of thousands of new oocysts in the intestines. Oocysts are distributed by faeces. Soon, they sporulate and become infective for chickens.

348. The intestinal lesions provoked by coccidia, are due to injury of the epithelial cells of the mucous coat where the parasites are developed and multiplied. The oocysts exist in the litter in premises and are distributed by clothes, shoes, dust, insects etc. Pathoanatomically, dehydration and a high degree of anaemia of the body and viscera are discovered.

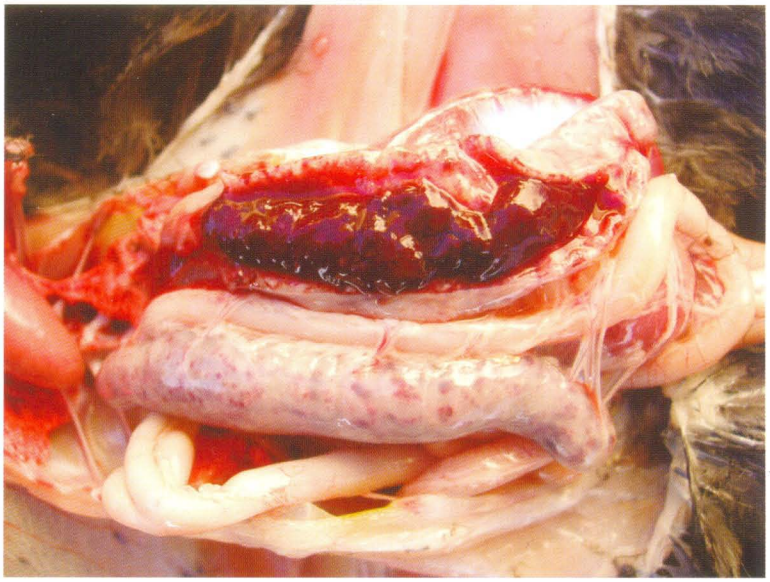
349. Anaemic appearance of internal organs.
The wet litter and the heat in premises favour the sporulation and therefore, the outbreak of coccidiosis.

350, 351. Depending on the localization of lesions in intestines, the coccidioses are divided into caecal, induced by *E. tenella*, and small intestinal, induced by *E. acervulina*, *E. brunetti*, *E. maxima*, *E. mitis*, *E. mivati*, *E. necatrix*, *E. praecox* and *E. nagani*. In caecal coccidiosis, a marked typhlitis is present and haemorrhages are seen through the intestinal wall.

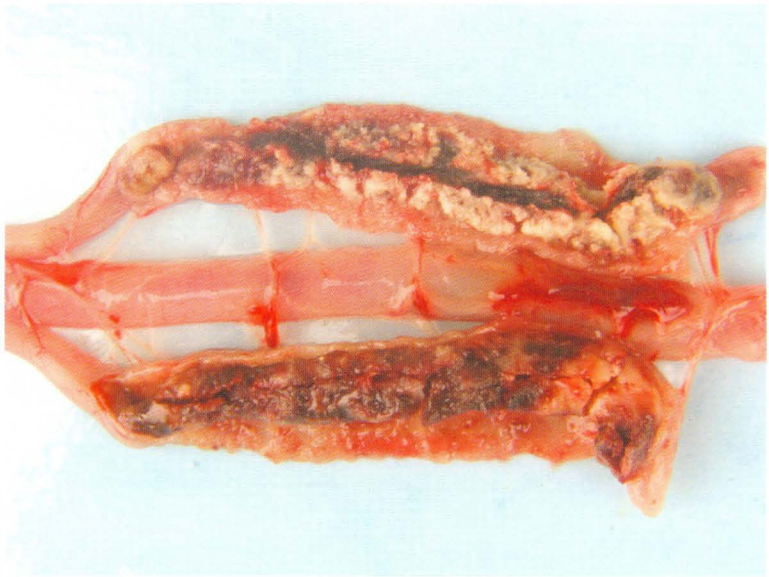


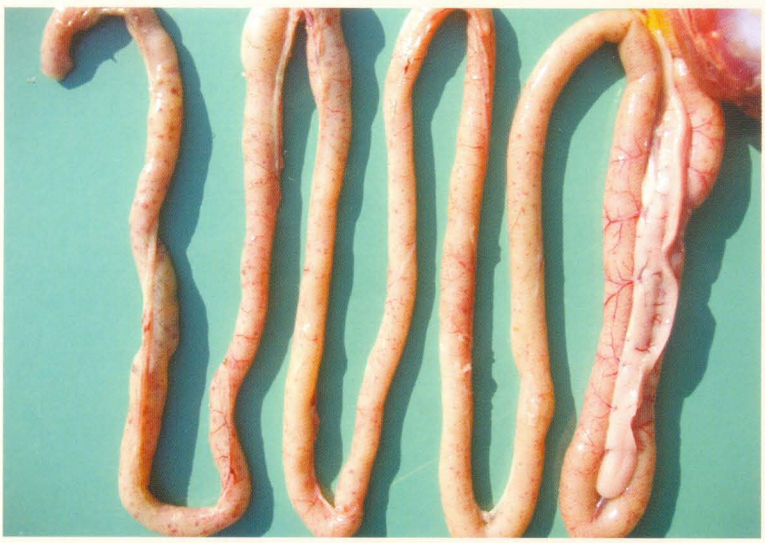


352. The caeca are filled with fresh or clotted blood.

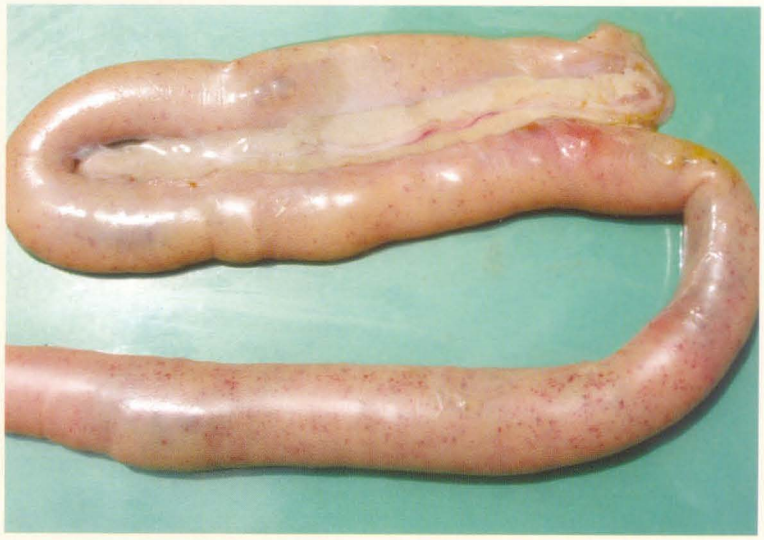


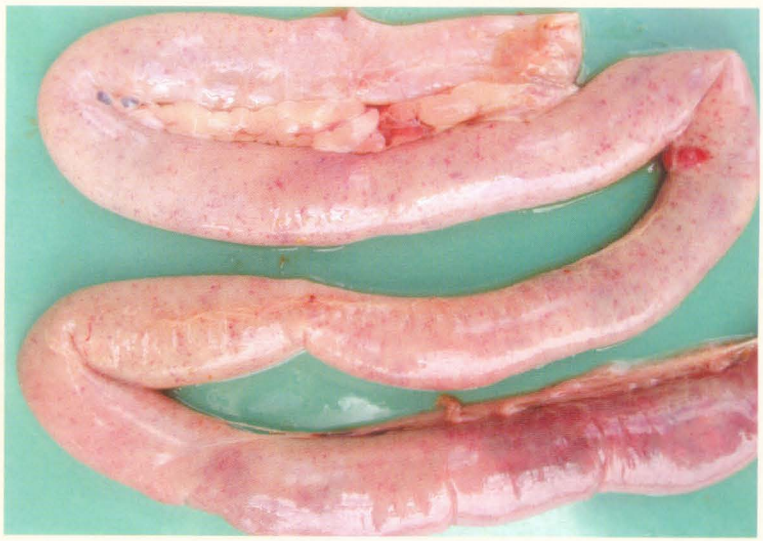
353. At a later stage, the caecal content becomes thicker, mixed with fibrinous exudate and acquires a cheese-like appearance.

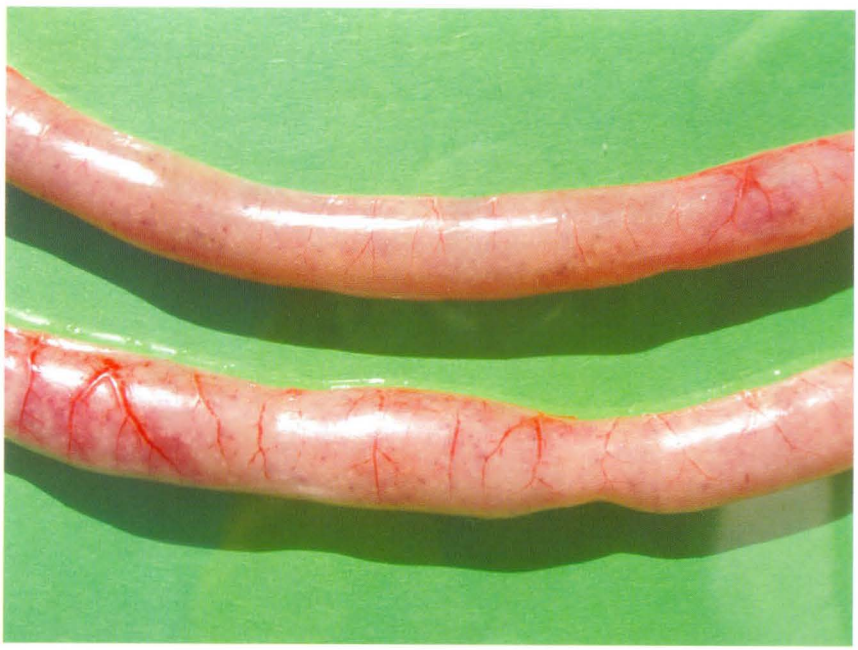




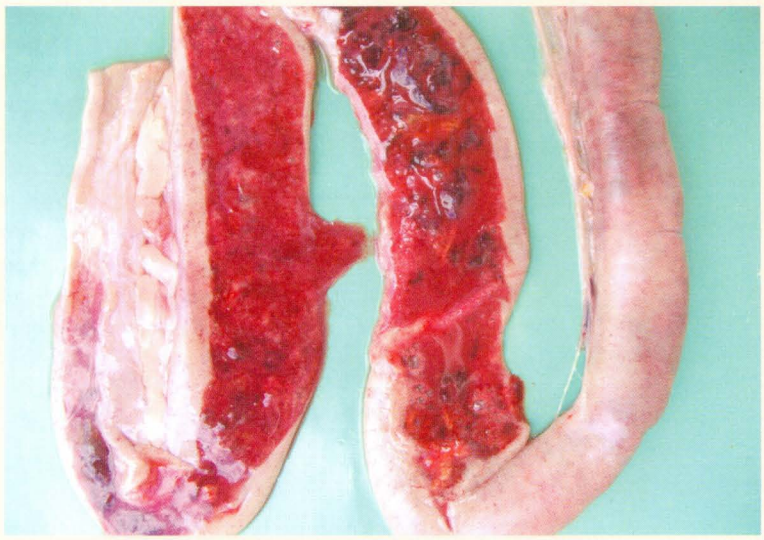
354, 355, 356. In small intestinal coccidioses, depending on the *Eimeria* species, haemorrhages with various intensities in different parts along the intestine are observed. In many instances, the haemorrhages are petechial and could be seen through the intestinal wall.

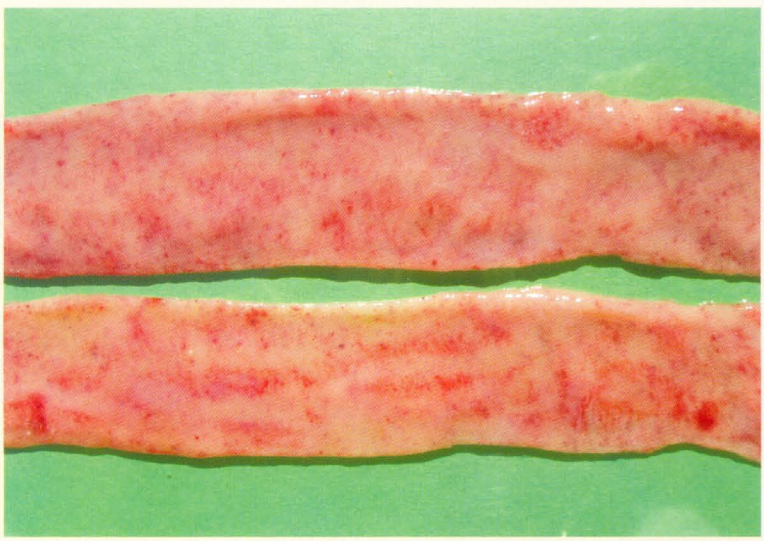






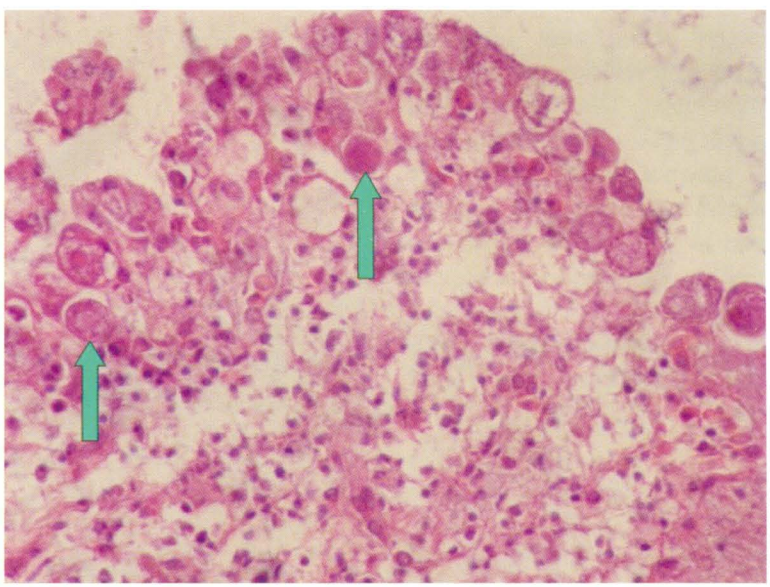
357. Sometimes, a reaction of the intestinal lymphoid tissue is present.

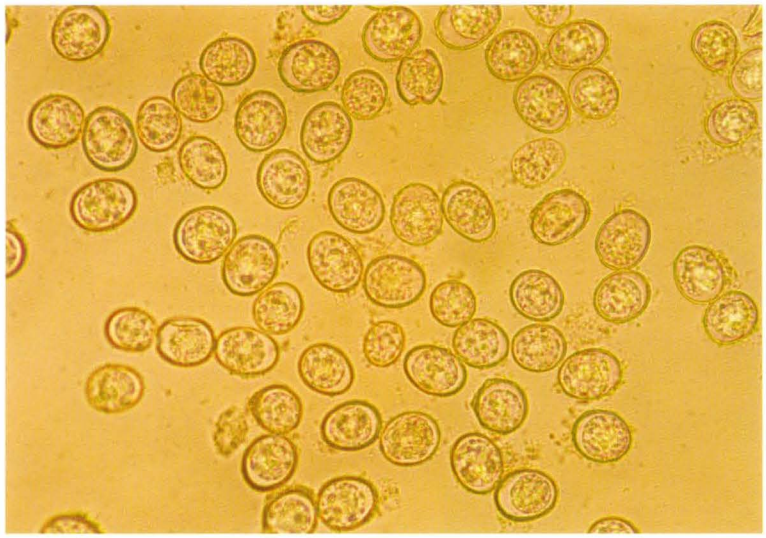




358, 359. The content is mixed with fresh or clotted blood, and the mucous coat is mottled with multiple petechial or larger haemorrhages.

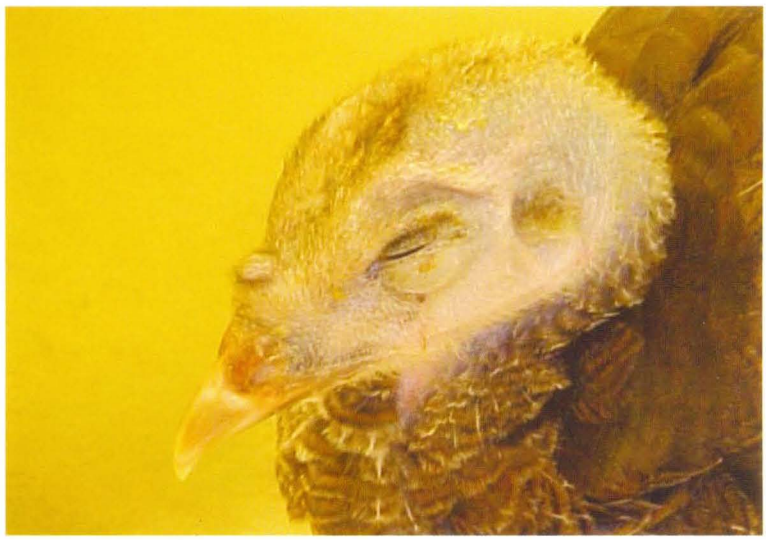
360. Histologically, Eimeria organisms at a various stage of development are detected in the epithelial intestinal cells. The **diagnosis** is made upon the results of the complex evaluation of the clinical picture, the macroscopic lesions, imprint preparations, histological study and flotation. Coccidioses should be differentiated from NE, UE and histomonosis (typhlohepatitis). **Treatment** - sulfonamides are widely used: sulfadimethoxine, sulfaquinoxaline, sulfamethazine, but they should not be used in layer hens. The supplementation of vitamins A and K promotes the recovery.





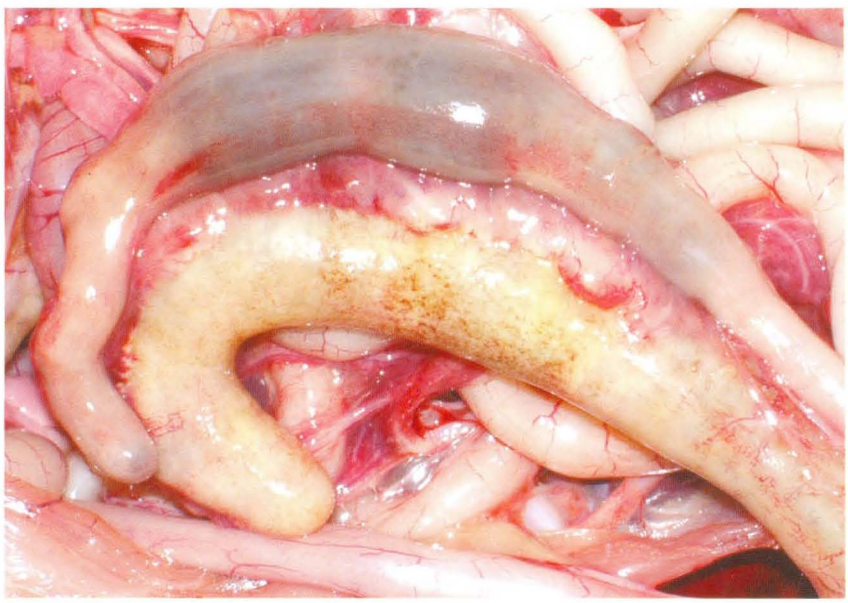
361. The microscopic examination of a native preparation of intestinal content or superficial mucosal layer reveals a significant number of oocysts in one observation field. **Prevention**. The use of coccidiostatics with forages on a rotation basis is the most extensively used means. The immunization against coccidiosis with commercial vaccines is used in broiler breeder flocks. If the chickens are exposed to the natural effect of a moderate number of oocysts in their environment, they develop immunity to the respective parasitic species.

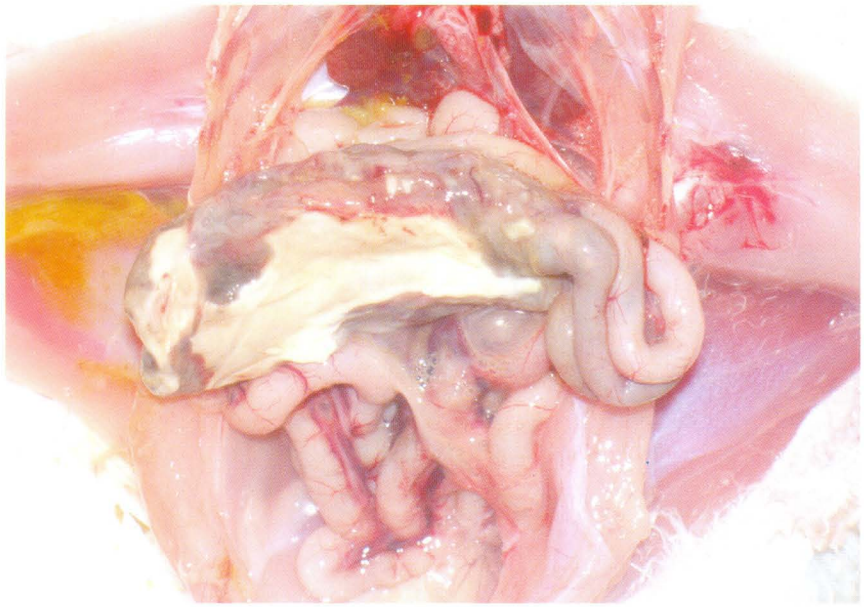
HISTOMONOSIS



362. The histomonosis is a protozoan disease, caused by *Histomonas meleagridis*, and characterized by necrotizing lesions affecting the liver and the caeca. Clinically, sulfur-yellow coloured faeces and depression are observed. A characteristic feature is the blackening of the skin of the head (blackhead), due to cyanosis.

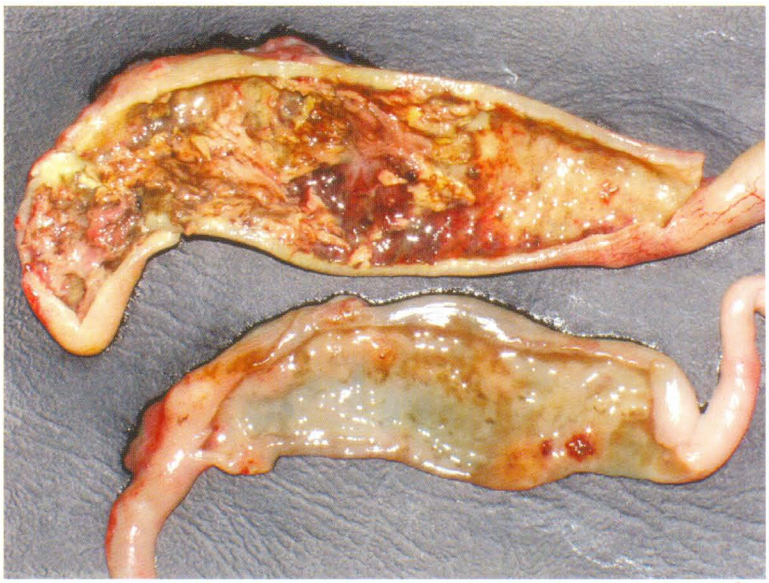
363. Pathoanatomically, bilateral enlargement of caeca with thickening of walls is observed. The aetiological agent is *Histomonas meleagridis,* a polymorphic flagellate that is present as flagellate in caeca and as amoeba in tissues. The trophozoites survive for several hours in the environment but in Heterakis eggs, they remain infective for more than a year.



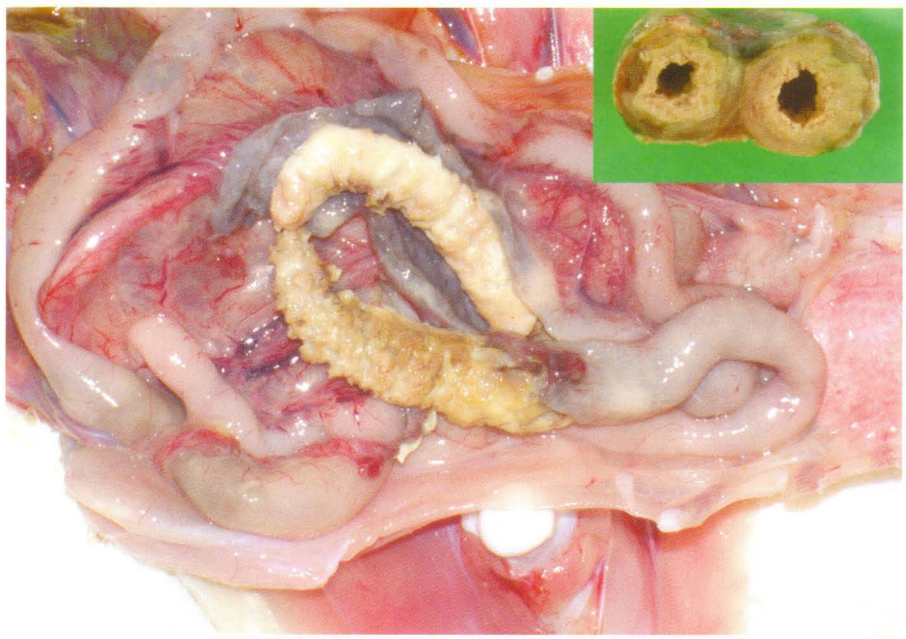


364. Often, the occurring typhlitis is the cause for adhesive peritonitis.

365. Susceptible species are turkeys, chickens, pheasants, rock partridges, guinea fowl, and geese. The turkeys are the most vulnerable between 3 and 12 weeks of age and chickens between 4 and 6 weeks of age. The caecal mucosa is usually ulcerated.

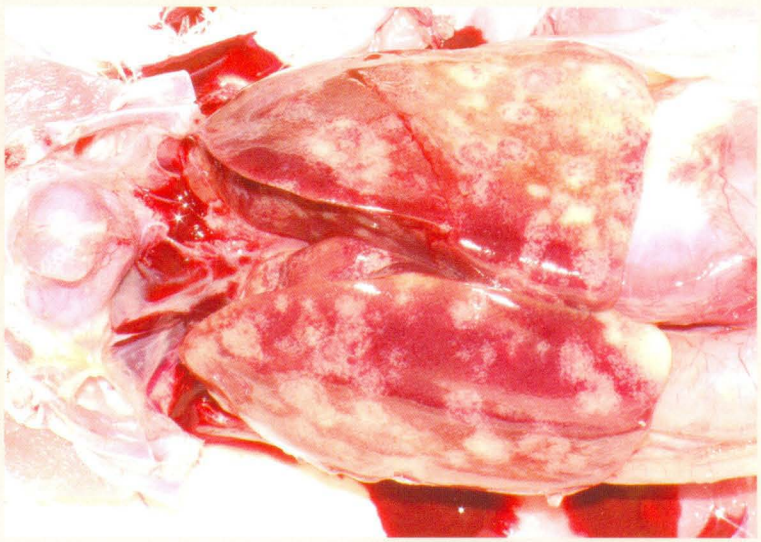


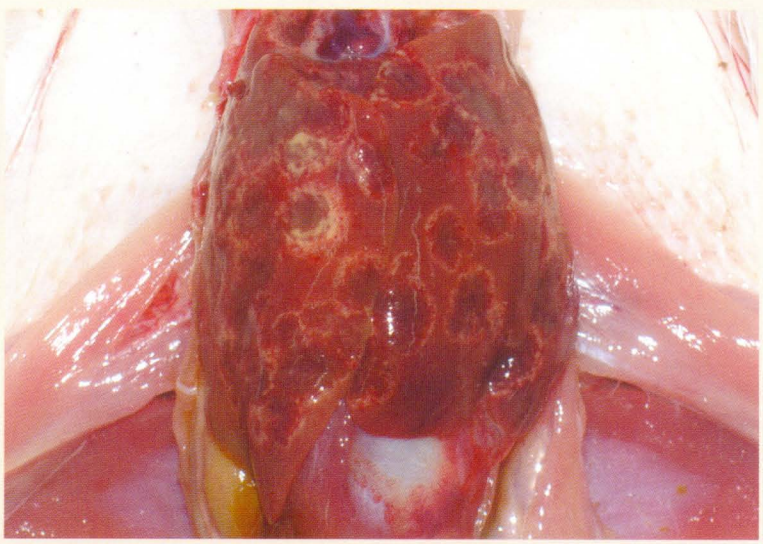
366. The main vector is *Heterakis gallinarum* through the eggs, respectively the larvae, where *Histomonas meleagridis* forms are found. Some wild birds could also serve as vectors. The caecal content is often mixed with blood.



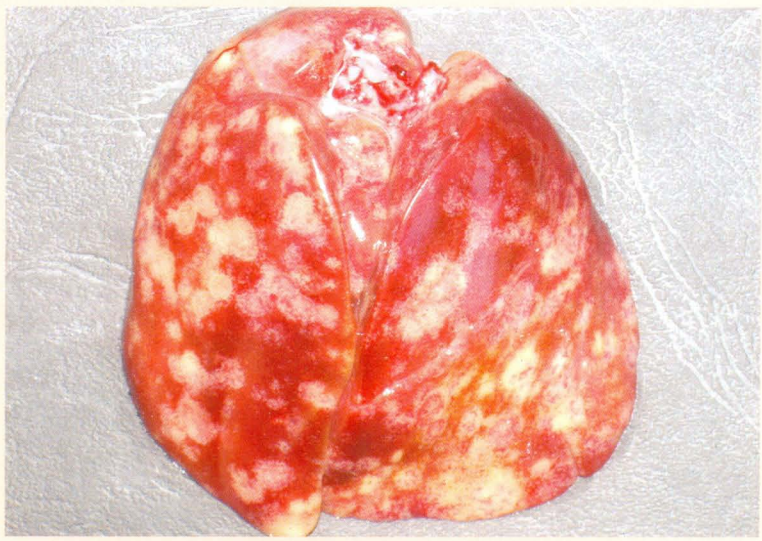
367. In older cases, crusts of dense caseous masses are formed into the caeca that thicken the intestinal wall and reduce the lumen (top right: transverse cross section through caeca).

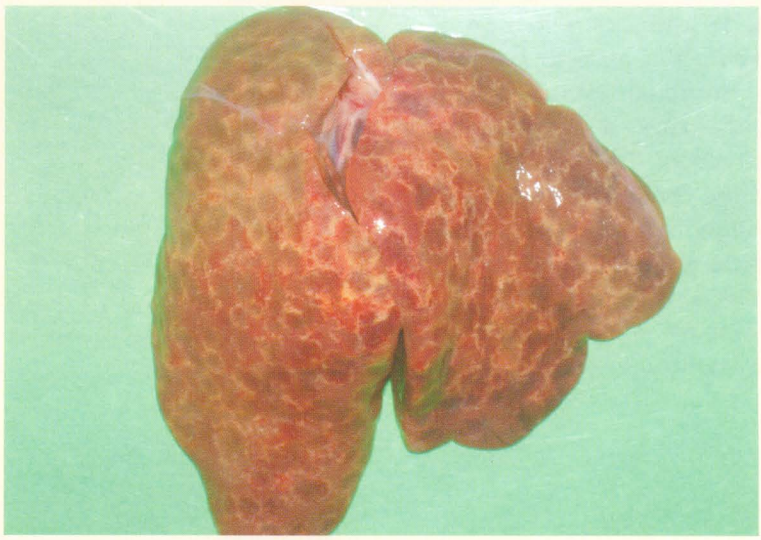
368, 369. Earth worms are mechanical vectors of *H. gallinarum* larvae. The main reservoirs of infection are hens and chickens. The morbidity rate amounts to 90% and the mortality rate to 70%. In the liver, irregularly outlined coagulation necroses with various size and colour, are observed.



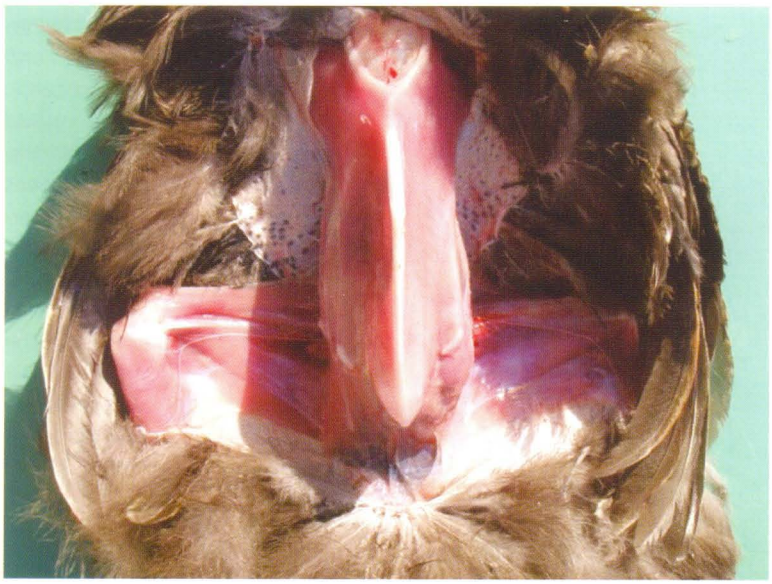


370, 371. Usually, necroses represent yellowish to grey or red (haemorrhagically infarcted), well delineated oci with diameter of about 1 - 2 cm. **Diagnosis** - it is made on the basis of the typical macroscopic lesions. When necessary, a histological study and phase-contrast microscopy of native preparations could be performed. Histomonosis should be differentiated from UE, coccidiosis and alimentary tract trichomonosis (*Trichomonas gallinarum*), where not counting the caeca, lesions are also present in the last third of small intestine.



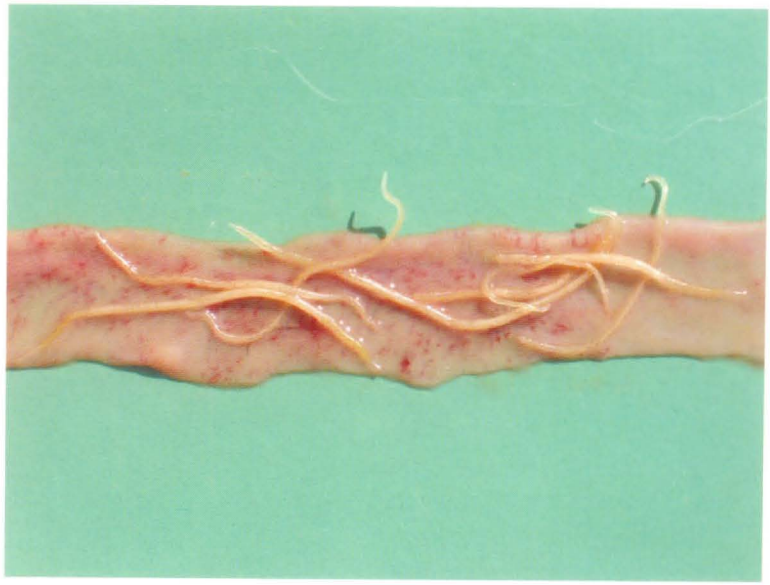


ASCARIDIOSIS

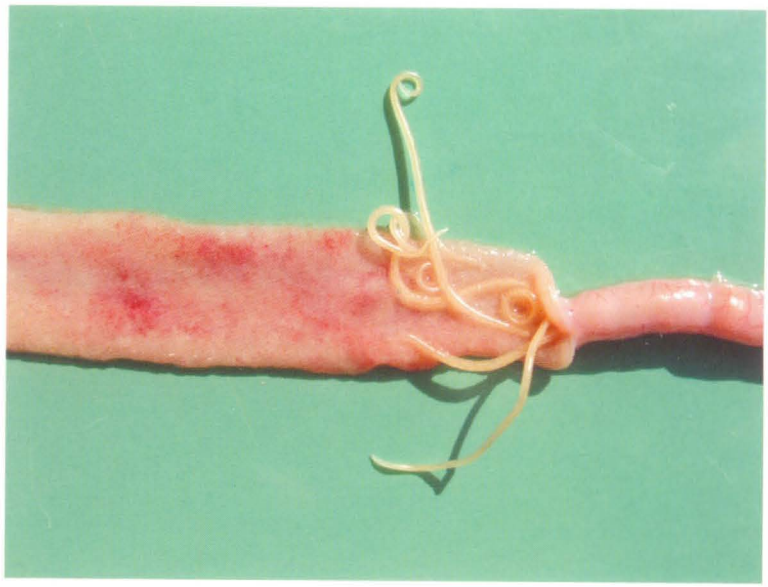


372. Ascaridiosis is one of the most prevalent helminthoses in fowl. It is caused by various species from the *Ascaridia* genus. The ascarids have a direct life cycle. Sometimes, it could involve paratenic hosts (earth worms). Infected birds are progressively emaciated, anaemic and sometimes diarrhoeic.

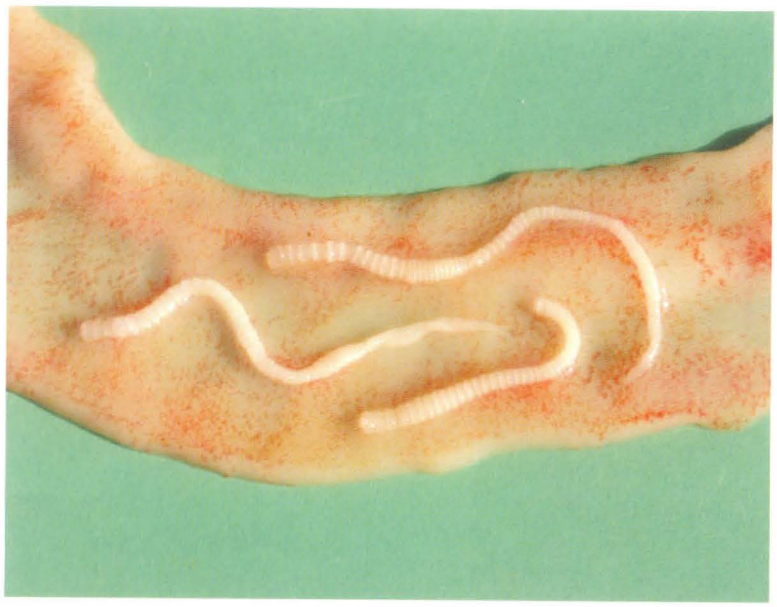
373. Pathoanatomically, haemorrhages of various intensities are found out in intestinal mucosa, catarrhal haemorrhagic enteritis and the parasites themselves are also observed.



374. In cases of extensive invasion, the ascarids block the intestinal lumen and could cause a complete obstruction. The treatment and the control are realized through regular pathoanatomical and coproovoscopic studies and performance of therapeutic and protective dehelminthizations.



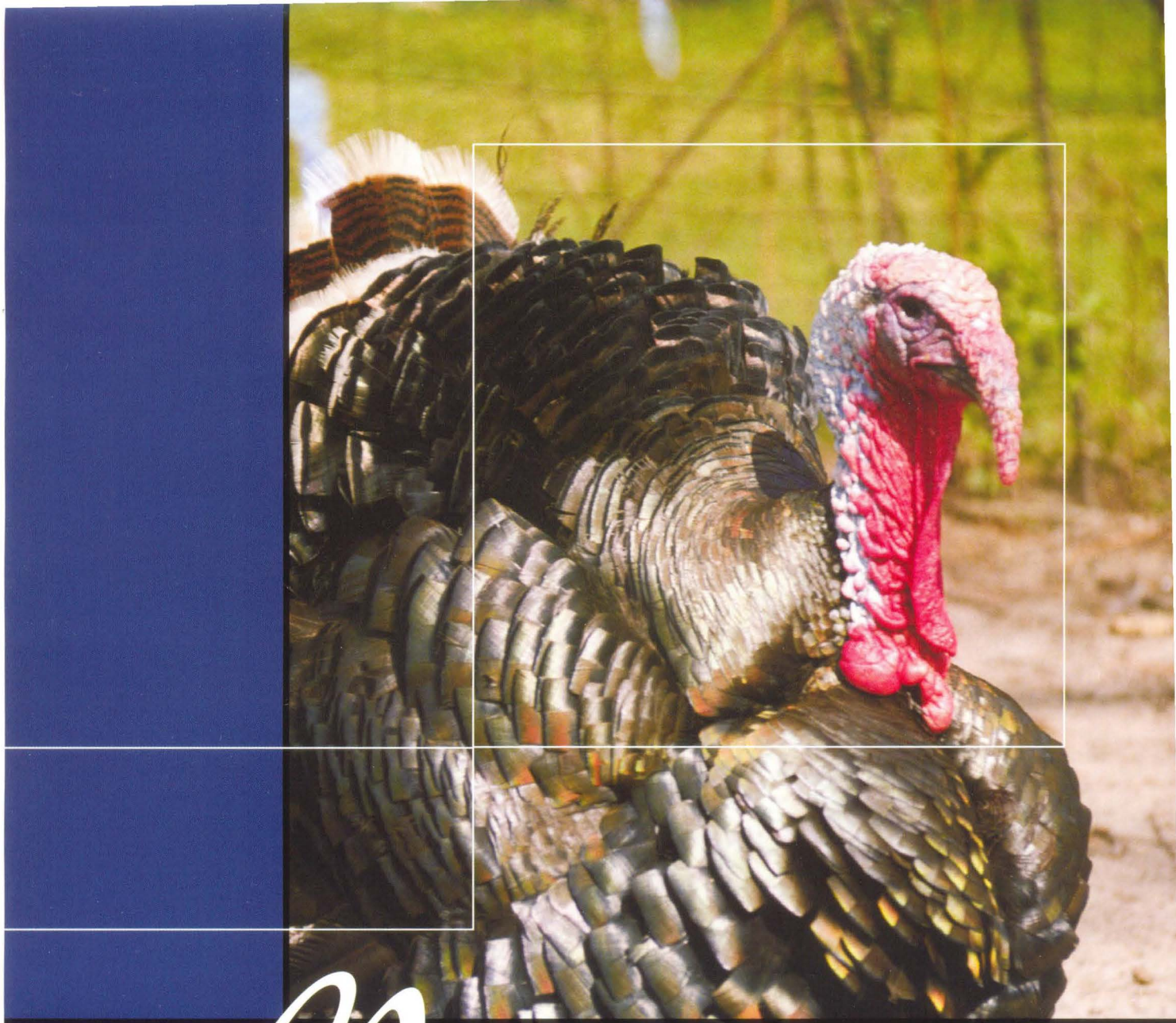
RAILLIETINOSIS



375. Raillietinosis is a cestodosis characterized by diarrhoea (sometimes bloody) during the acute stage and emaciation to cachexia and anaemic during the chronic stage. It is caused by some representatives of the *Raillietina* genus that parasitize in various areas of the small intestine. The usual intermediate hosts are ants or other insects. Pathoanatomically, haemorrhages with various intensities in the intestinal mucosa, catarrhal haemorrhagic enteritis and the parasites themselves are found out throughout the gross examination. The treatment and the control are done by dehelminthization of all birds in the affected farm.

KNEMIDOKOPTOSIS

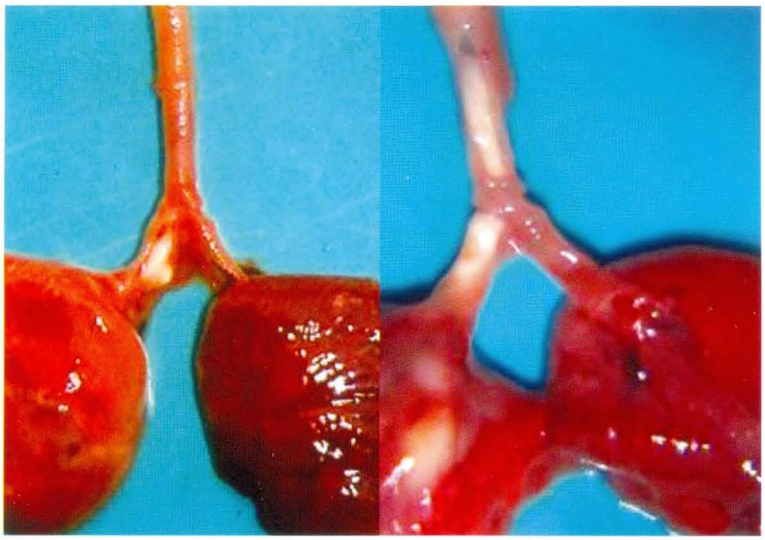
376. Knemidokoptosis, known also as scaly or chalky legs, is characterized by the appearance of extensive, rough and hard crusts on the featherless part of the legs. Adult birds are generally affected (hens, turkeys, pheasants, exotic birds). It is caused by Knemidokoptes mutans. The lesions are secondary to an inflammatory reaction, in which the thickening of the cutaneous epidermis together with released exudate forms the crusts on legs. The infection occurs by contact between birds. The control is done by isolation of affected birds, leg bathing with warm acaricide solution and warm vegetable oil that helps the decrustation.



Mycoses and mycotoxicoses

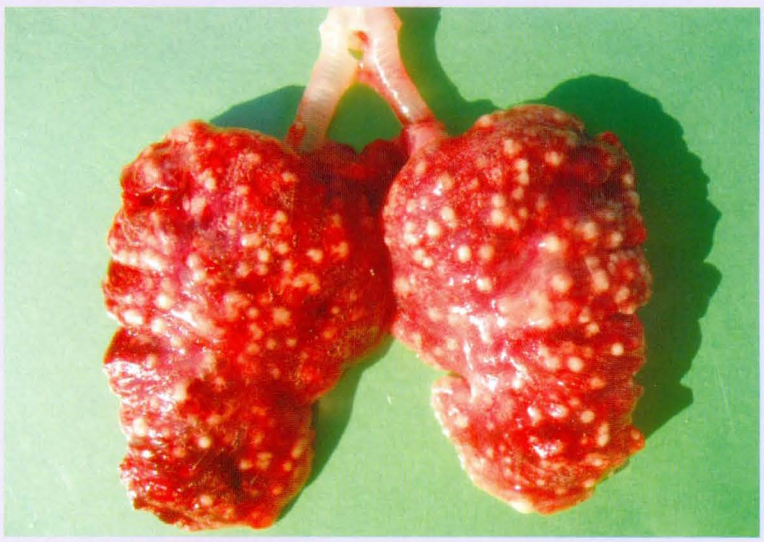
MYCOTIC DISEASES
ASPERGILLOSIS

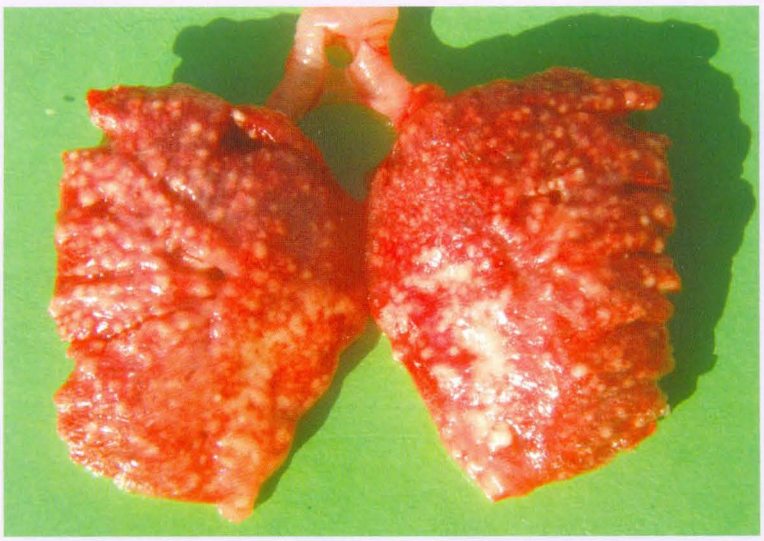
377. Aspergillosis is an acute or chronic respiratory disease. In rare instances, peritoneal, visceral or systemic lesions could be observed. It is caused by *Aspergillus fumigatus*. Dyspnea, enhanced, tense and heavy breathing are observed. Sometimes, rales and cyanosis could occur.

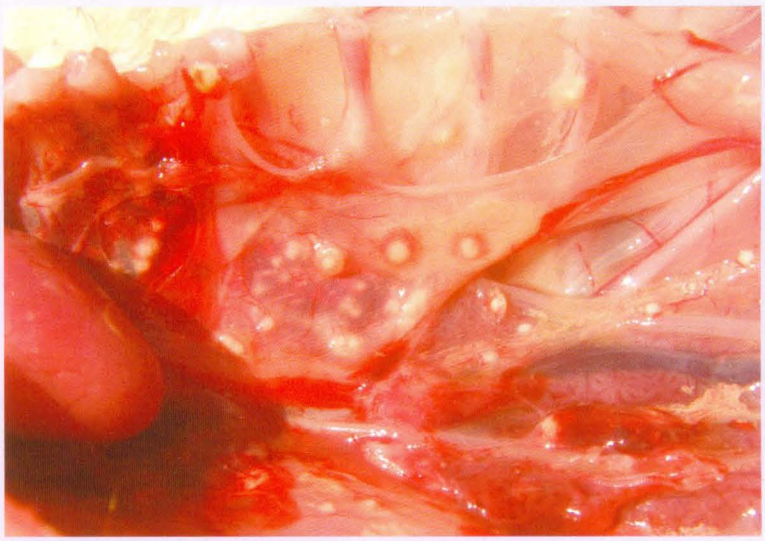


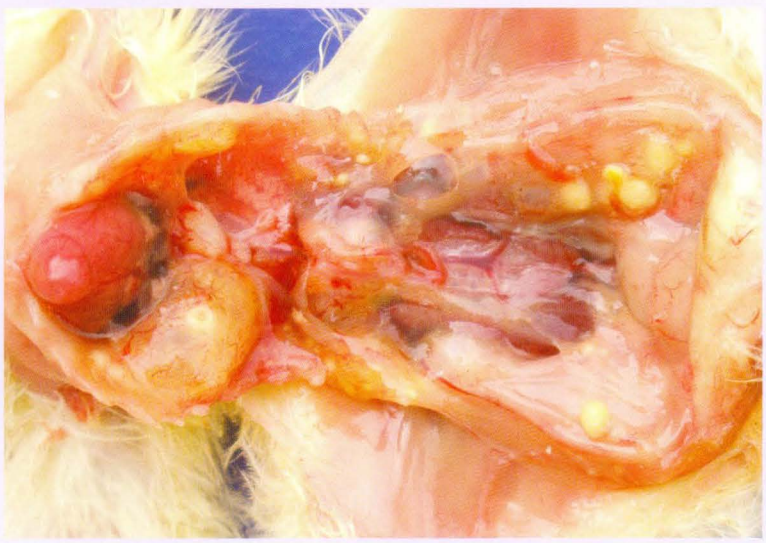
378. In the acute form, a serous fibrinous pneumonia is observed. Within the trachea and the main bronchi, usually near the bifurcation, obturation masses of coagulated fibrinous exudate are detected.

379, 380.
In the nodular form, multiple grey whitish or yellowish dense nodes in the lungs are observed (379 and 380). Chickens, turkeys, waterfowl and many other domestic or wild fowl are susceptible.

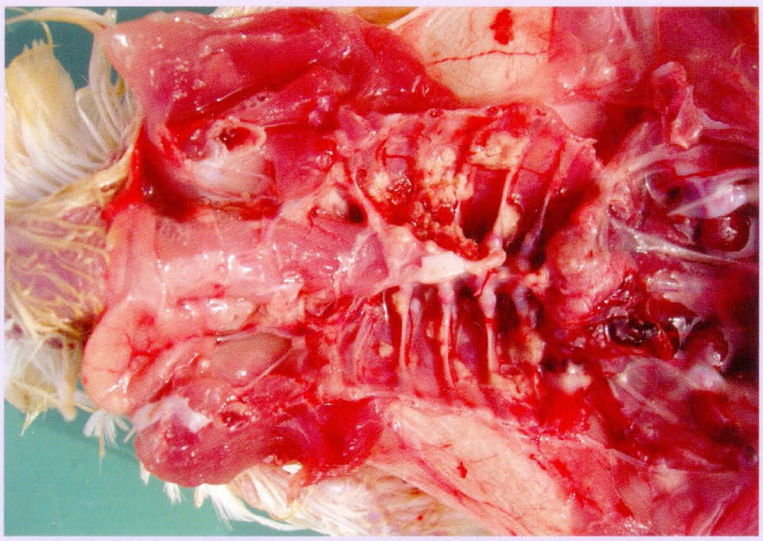


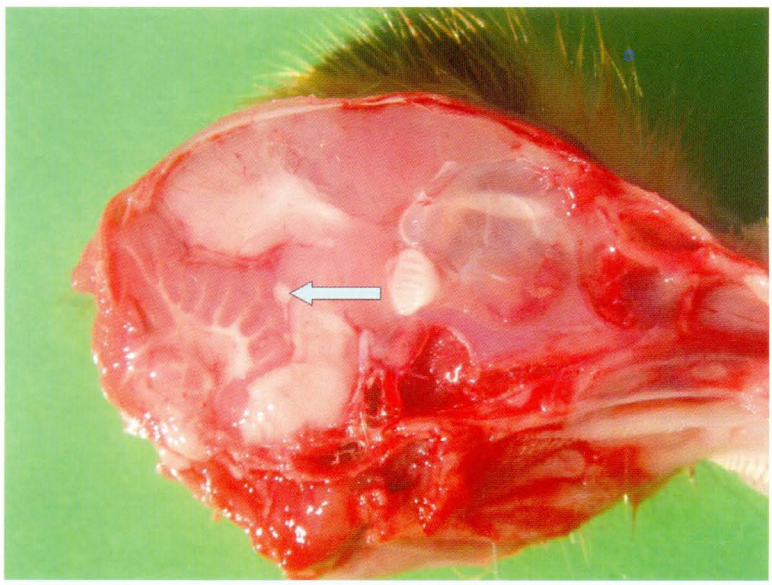






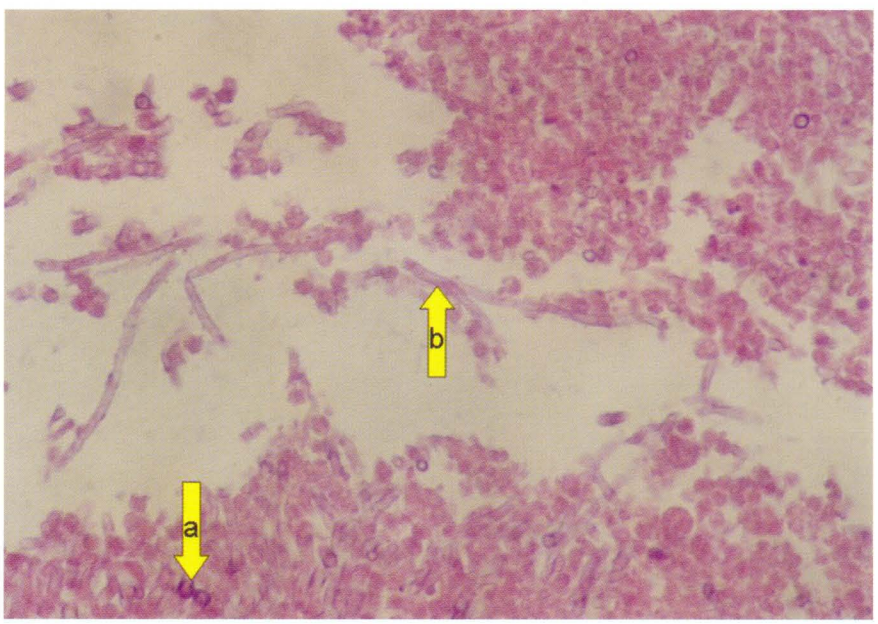
381, 382, 383.
Aspergillus granulomas of the serous coats: pectoral (381), abdominal air sacs (32) and pleural (383), are sometimes compressed. The infection could occur inside (incubatory aspergillosis)or outside the hatchery. *Aspergillus fumigatus* penetrates through the egg shell under ideal conditions for development and infects the embryos. The eggs explode and release a significant number of spores that contaminate the hatchery and the environment. The spores are spread via the ventilation system that results in severe outbreaks in chickens younger than 3 weeks. Outside the hatchery, the infection occurs by inhalation of numerous spores from the contaminated forage, litter or environment. The overcrowding and the increased humidity are prerequisites for outbreaks.

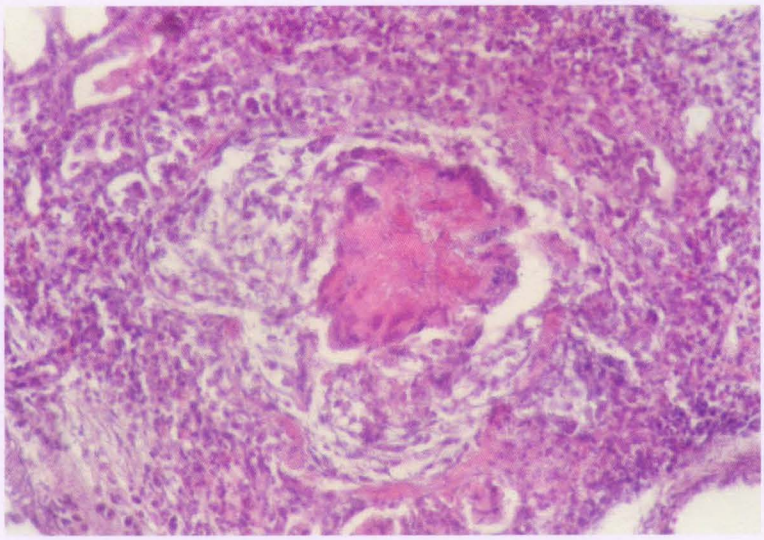


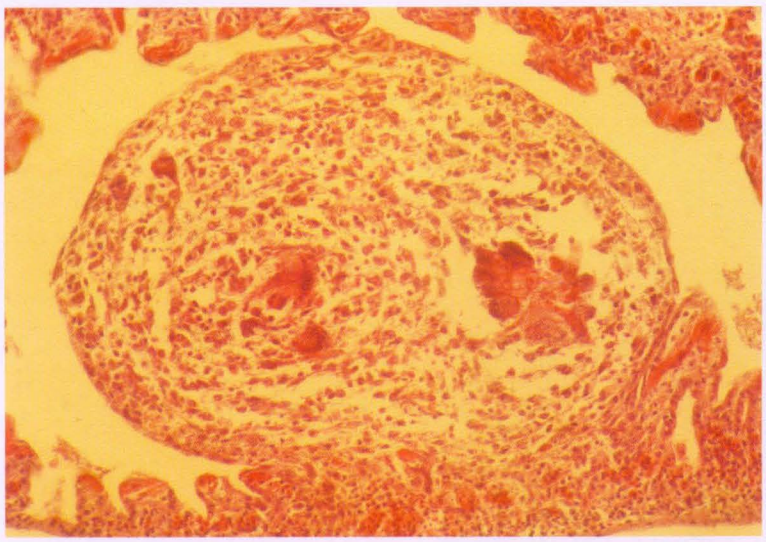


384. Rarely, as a systemic manifestation, *Aspergillus* granulomas in the brain could be detected, when spores are transported via the blood circulation.

385. The signs and macroscopic lesions are very indicative for aspergillosis. They could be confirmed by histology too. In acute aspergillosis, among the inflammatory necrotic masses, the spores are observed (arrow **a**) as well as the grown hyphae (arrow **b**) of the mould.







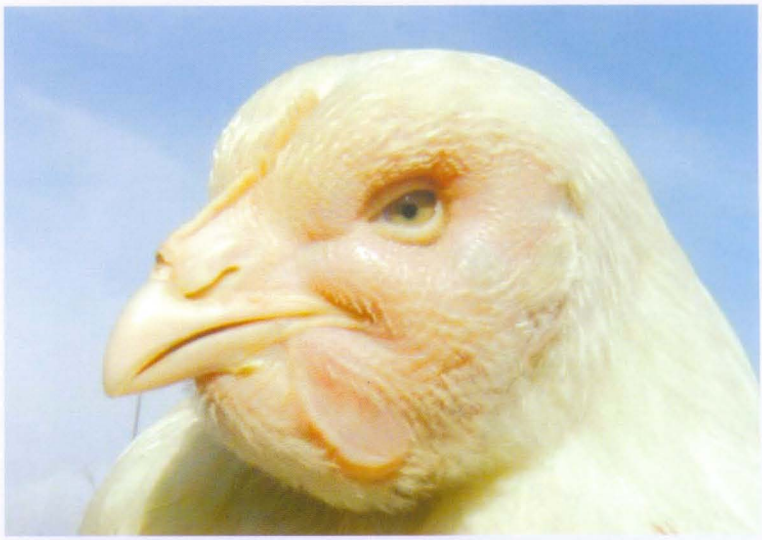
386, 387. In the nodular form, a characteristic *Aspergillus* granulomatous structure of tissues is discovered (386 and 387). The **control** includes: collection of clean eggs for hatching, disinfection and fumigation of eggs, incubators and hatcheries, regular checking of ventilation systems and change of air filtres in hatcheries, monitoring of hatcheries and the environment for mould contamination, use of dry and clean litter and non-contaminated forages, optimization of aeration and the humidity in premises where birds are housed. The **treatment** with fungicide antibiotics and different antimycotic drugs is not always efficient. Nistatin and copper sulfate (1:2000) with drinking water could be used.

388. The mould is developed after inoculation of material on an appropriate nutrient media.

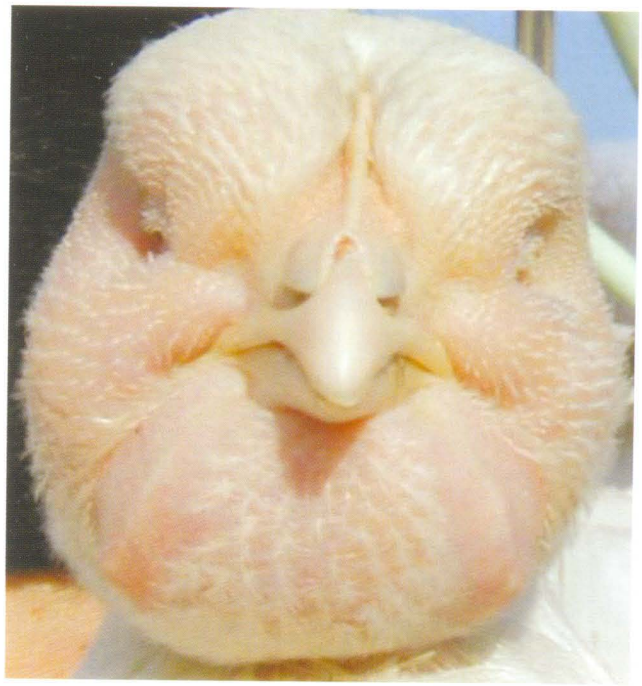
ASPERGILLUS GRANULOMATOUS DERMATITIS

389. The *Aspergillus* granulomatous dermatitis as a postvaccinal complication is observed in growing broiler breeders. Following subcutaneous application of oil adjuvant vaccine in the lower part of the neck, a marked local reaction with extensive swelling of the head is observed.

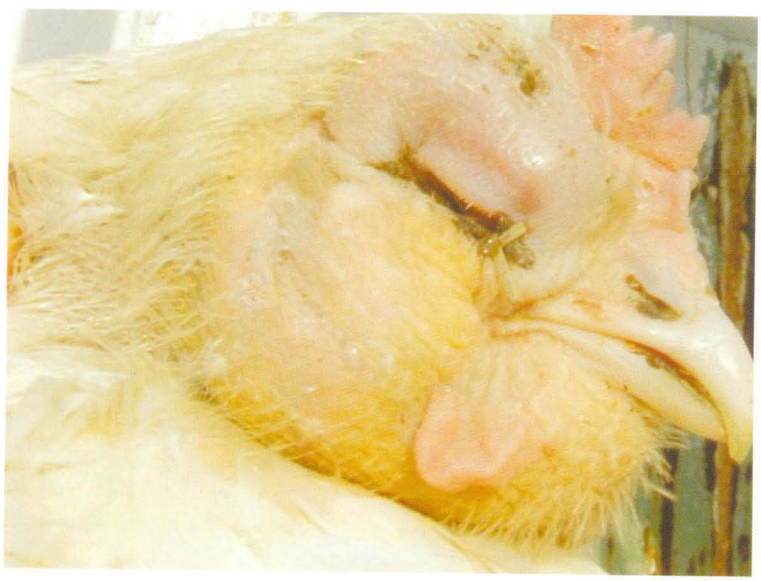
390, 391. The swellings involve the subcutaneous tissue of periorbital sinuses, the mandibular space, even the entire skull without the skin adnexa.



392. The head is ballooned with strongly stretched skin.

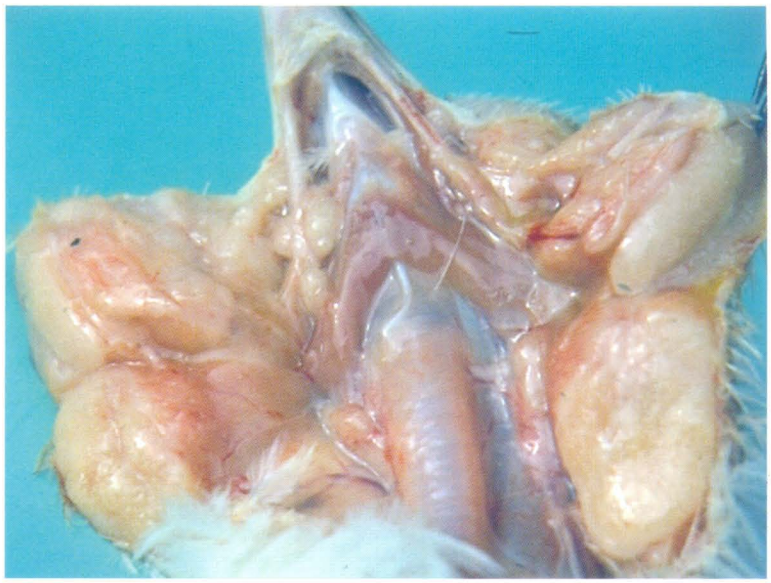


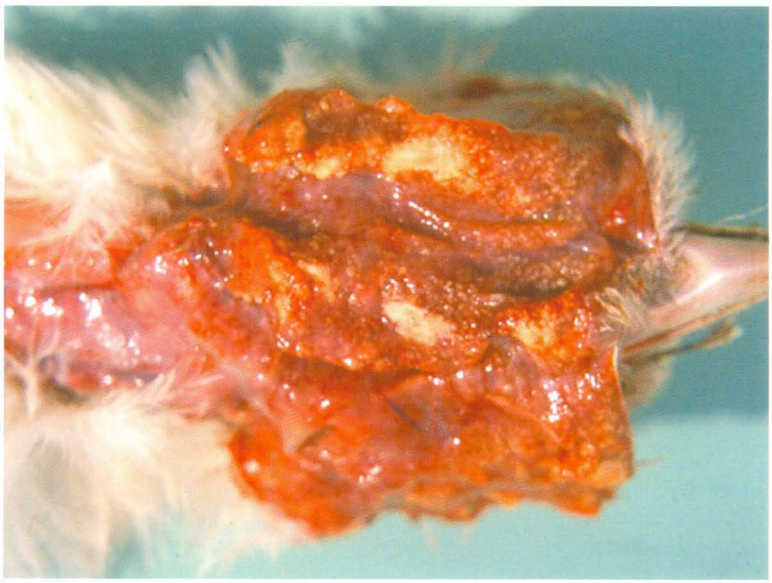
393. In some cases, the covering skin acquires a blue-greenish colour.



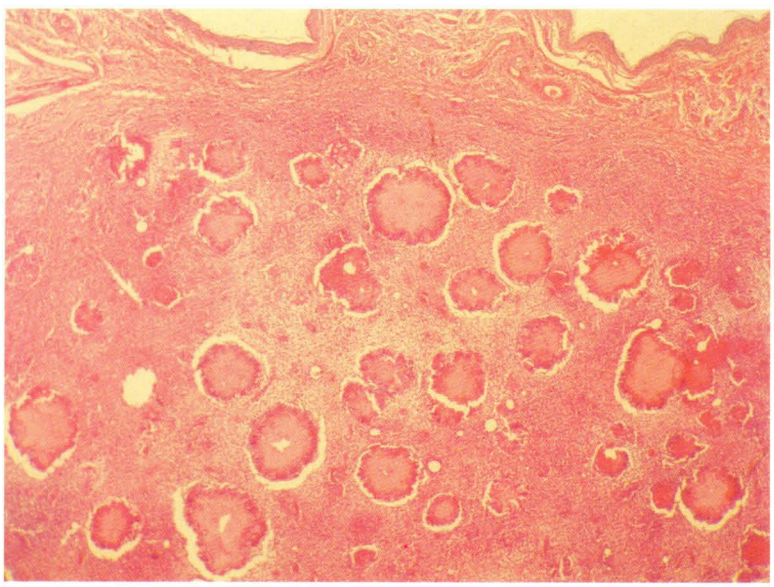
394. The eyeball is sometimes affected by necrotic colliquation.

395. Among the cross sectional surface if the highly oedematous subcutaneous tissue, multiple structures resembling sand grains are detected.

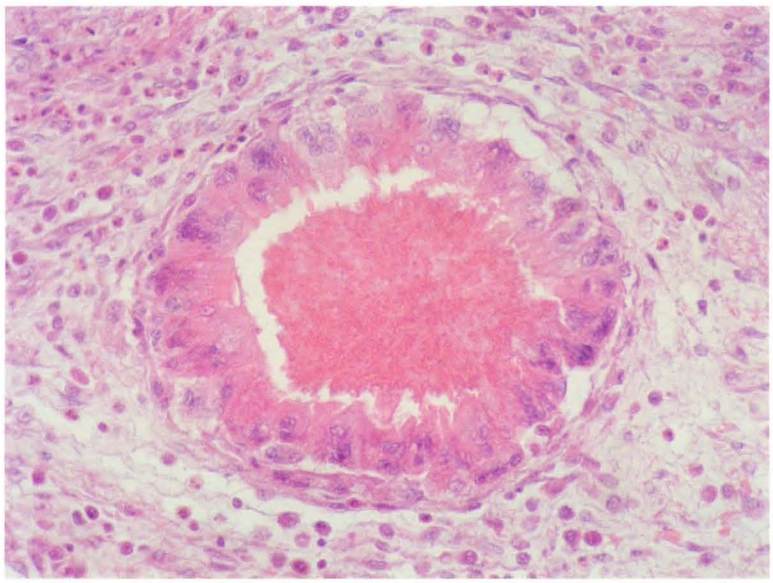




396. At a later stage, a spontaneous regression of oedemas occurs, but granulomatous formations continue to be visible in the subcutaneous tissue.

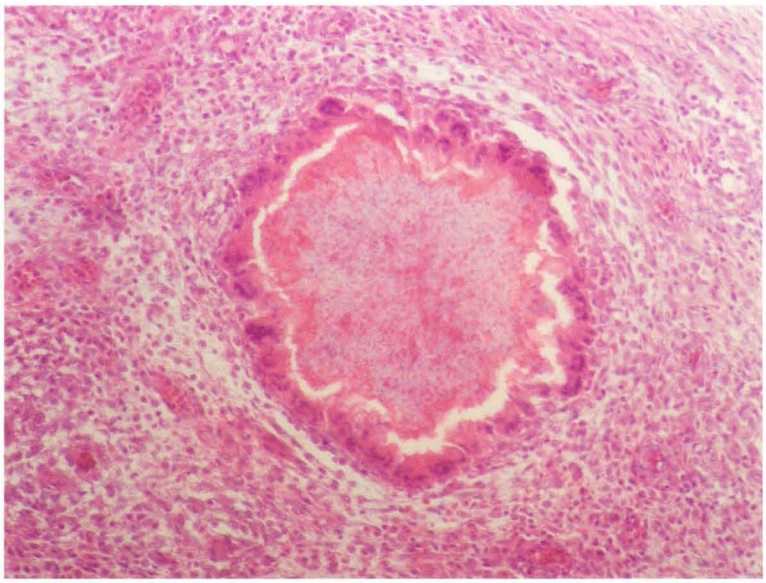


397. Histologically, among the oedematous subcutaneous connective tissue, multiple granulomas are detected.

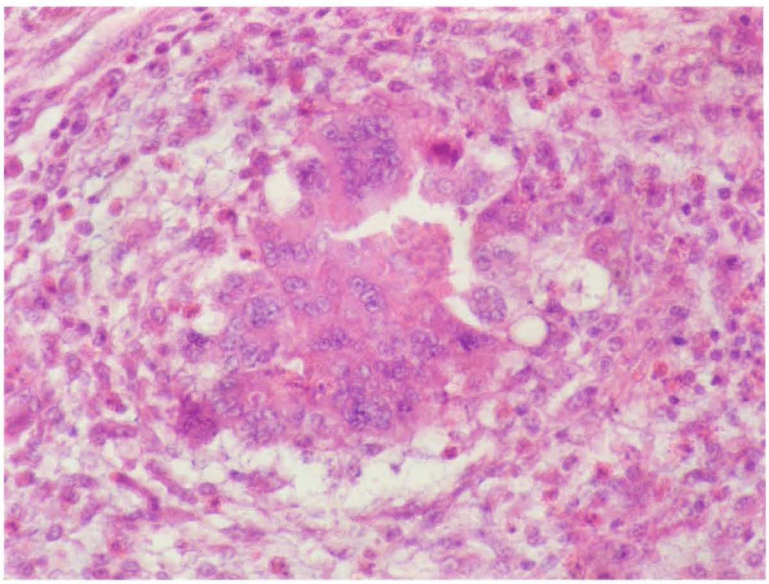


398. The central zone of granulomas is an inflammatory necrotic detritus with many eosinophilic leukocytes delineated within.

399. Peripherally, as a crown, multinuclear giant cells of a foreign-body type are arranged, with fibroblast fibrocyte growths around them.

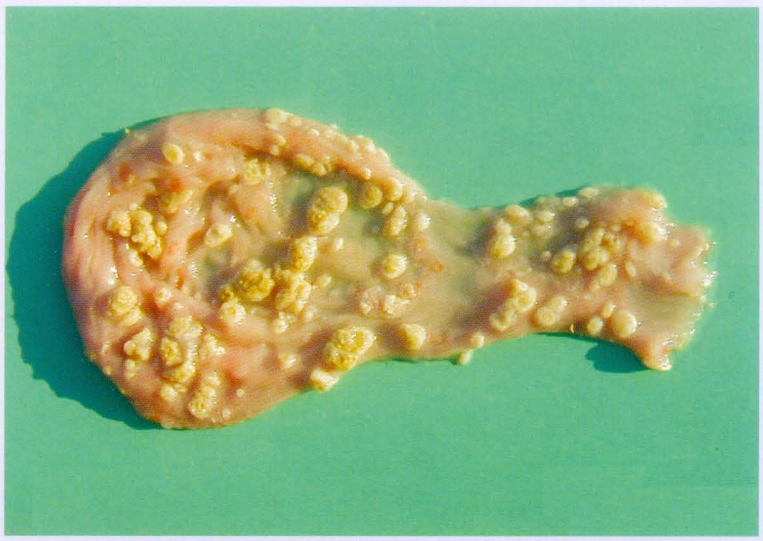


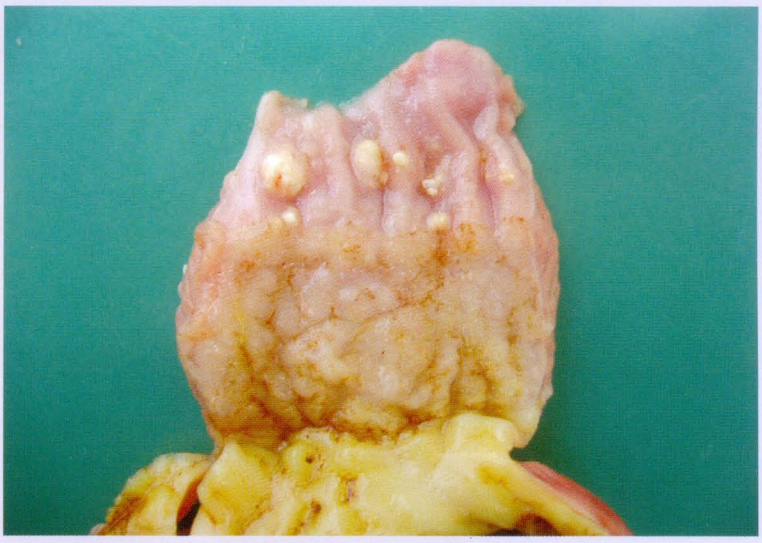
400. At a later stage of the condition, after phagocytosis of the detritus, the structure of granulomas is mainly composed by foreign-body giant cells and connective tissue growths.



CANDIDIASIS

401, 402. Candidiasis is an alimentary tract disease, caused by the fungal yeast *Candida albicans* and usually occurs as a secondary infection. Predisposing factors are the lack of good hygiene, prolonged antibiotic treatment, vitamin deficiency, severe parasitic infections and immuno-deficiencies. The production of a soluble endotoxin is also contributing to the pathogenicity of the agent. The lesions are usually detected in the crop, lips, oesophagus, but could affect the proventriculus and more rarely, the intestines. The affected mucosa is diffusely or focally thickened, raised and wrinkled, white, resembling a towel. Possibly, the lesions could look as pseudomembranous or diphtheroid coatings. The histological study confirms the **diagnosis** by showing the fungal hyphae in the affected mucosa. Usually, a 1:2000 copper sulfate solution is used with drinking water for **prevention** and **control**. The application of nistatin in water or forage is efficient against candidiasis in turkeys.





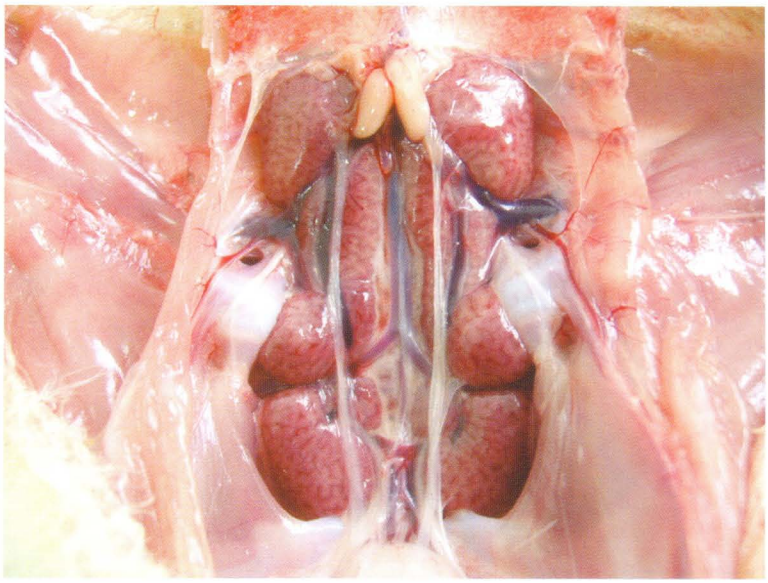
MYCOTOXICOSES

AFLATOXICOSIS

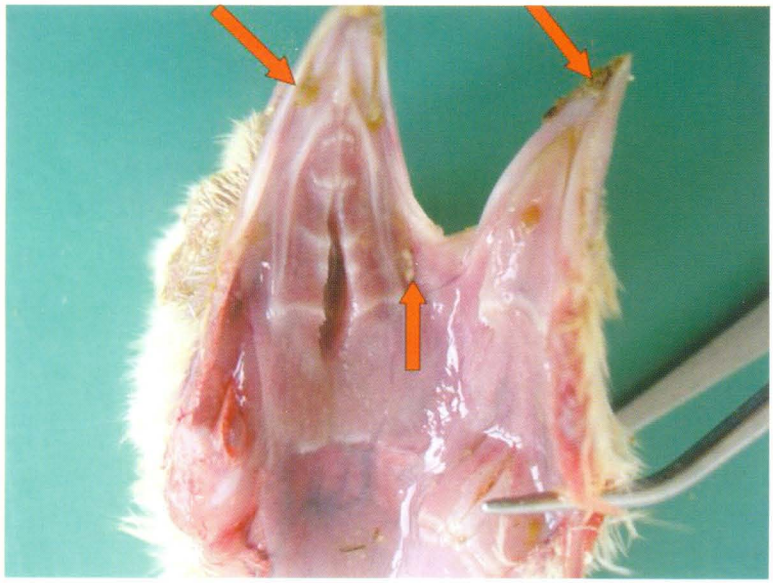
403. Aflatoxins are very toxic and carcinogenic mycotoxins, produced by moulds of the *Aspergillus* and *Penicillium* genera. In broilers, paralysis and lying down could be observed. The growth of affected birds is retarded.

404, 405, 406.
A more typical gross finding is the yellowish to yellow-earth colour of the liver, the multiple haemorrhages and a characteristic reticular appearance of the capsular surface.

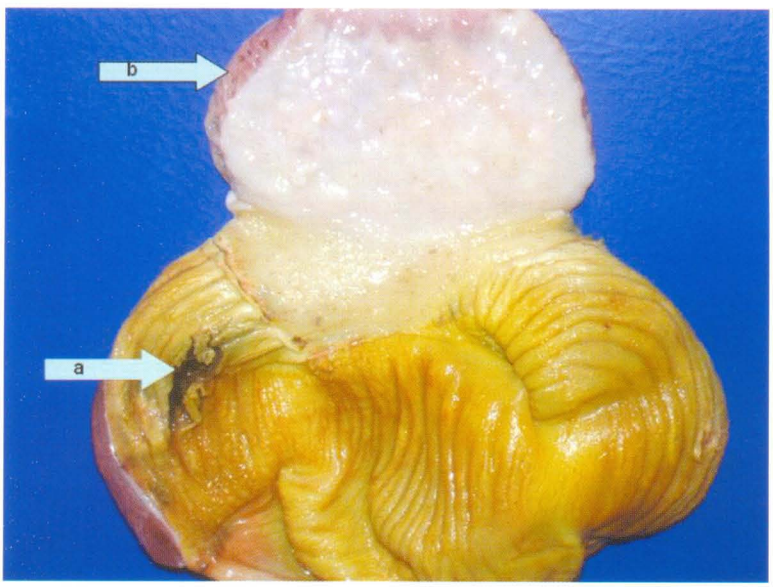
407. In severe intoxications, the kidneys are enlarged and filled with urates.



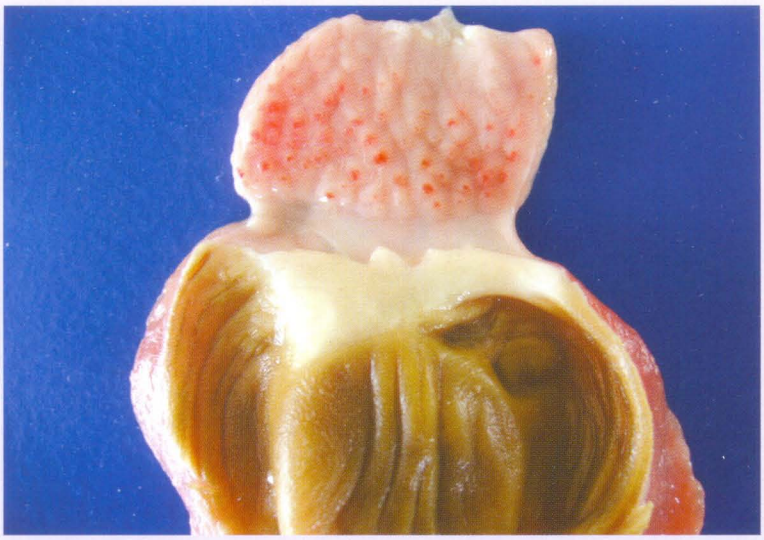
FUSARIOTOXICOSES



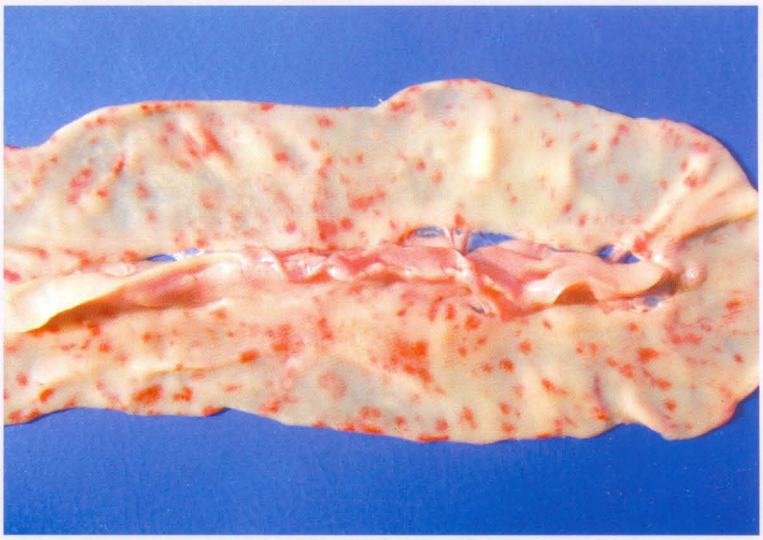
408. The *Fusarium* genus produces numerous mycotoxins, out of which, the most important for poultry pathology are trichothecenes, fumosinins, moniliformin, fusarochromanone and zearalenone. The *Fusarium* toxins possess a pronounced caustic effect, resulting in necroses and crusts of the buccal mucosa.

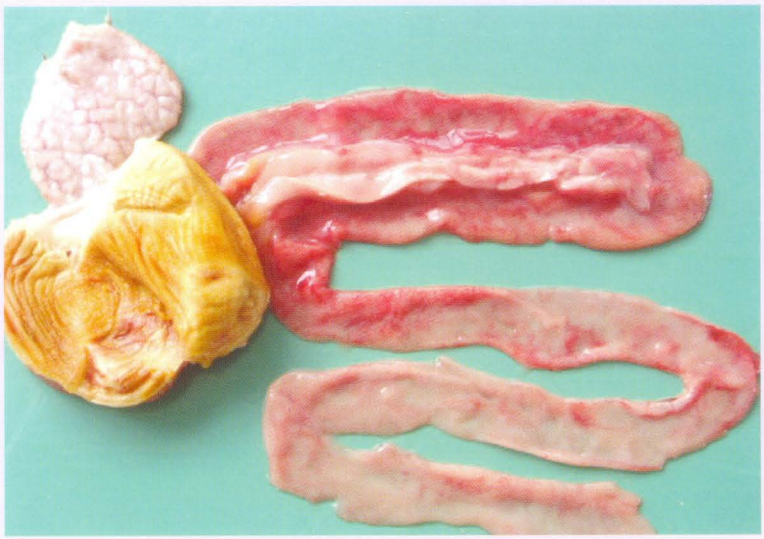


409. The caustic effect of some fusariotoxins is the cause of the commonly detected erosions and ulcers in gizzard cuticulum (arrow **a**). Note the thickened wall of the proventriculus (arrow **b**).



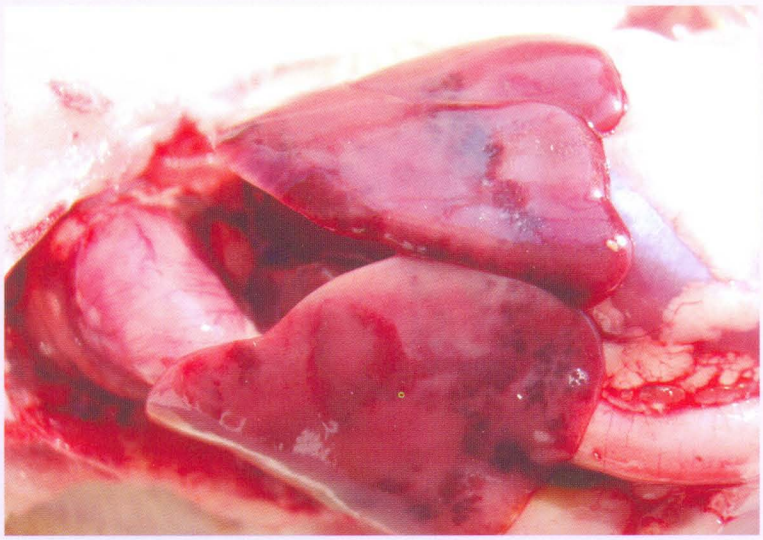
414, 415, 416.
The lesions include also reddening and hemorrhage of intestinal mucosa. The size of hemorrhages varies from 23 to 56 mm in diameter and they could often be seen through the intestinal wall. Usually, the mucous coat of the duodenum and the initial part of the ileum are affected.

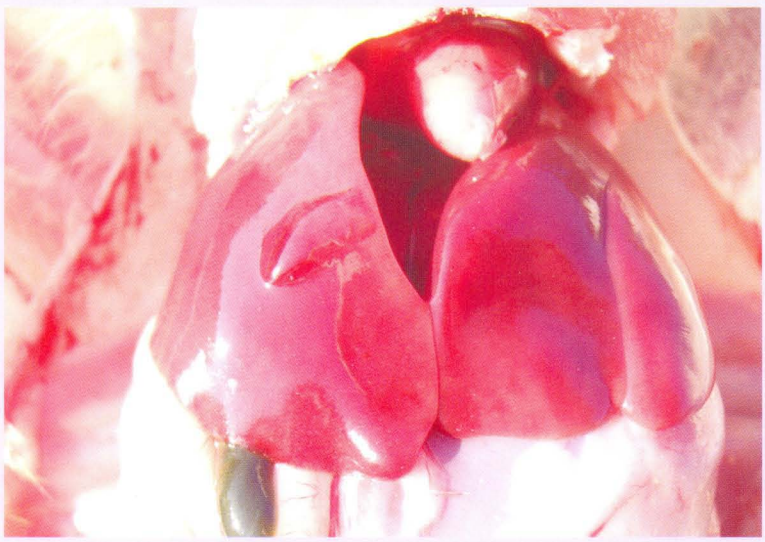




417. A significant part of dead bodies are dehydrated.

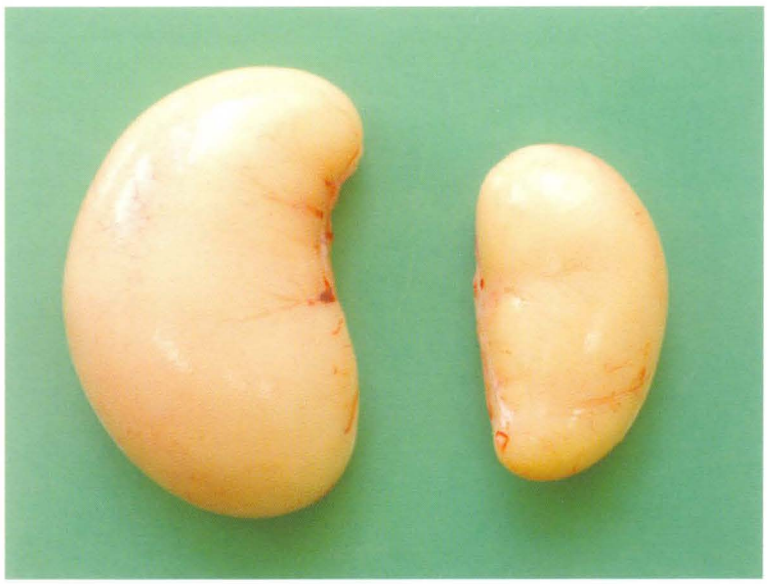
418, 419. Inside the liver, hemorrhages with various intensities, outlined at the background of severe dystrophic changes, are detected.

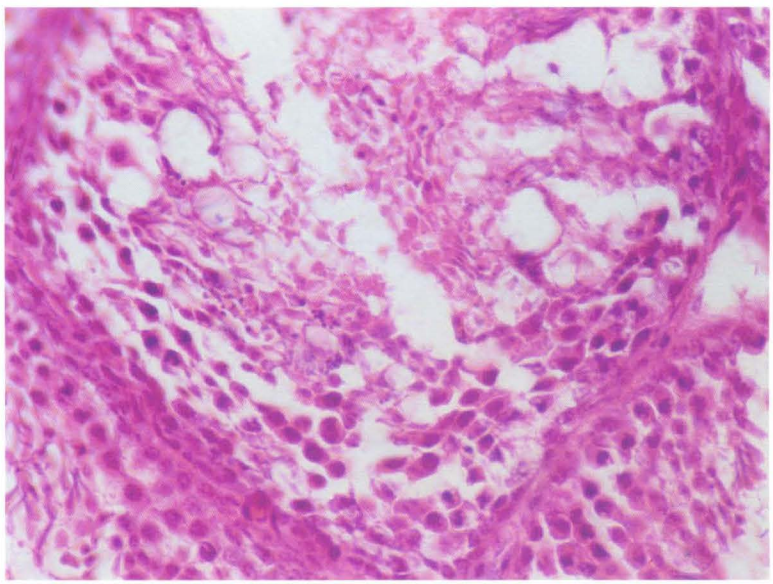




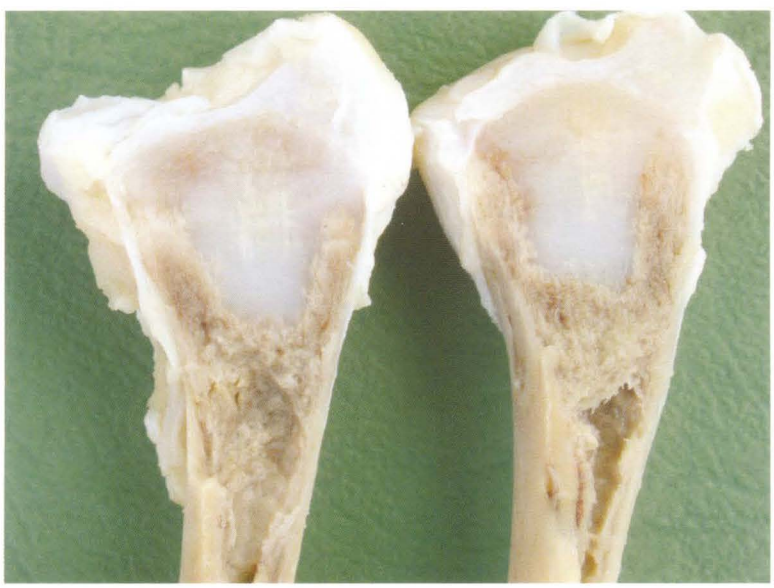
420, 421. Frequent findings in fusariotoxicoses are the massive subcapsular liver haematomas, causing sudden death in broilers.

422. The fusariotoxin zearalenone has an effect, identical to that of oestrogenic hormones and results in reduction of testes in cocks. Left - normal; right - atrophied testis in a cock, in whose diet high zearalenone concentrations have been determined.



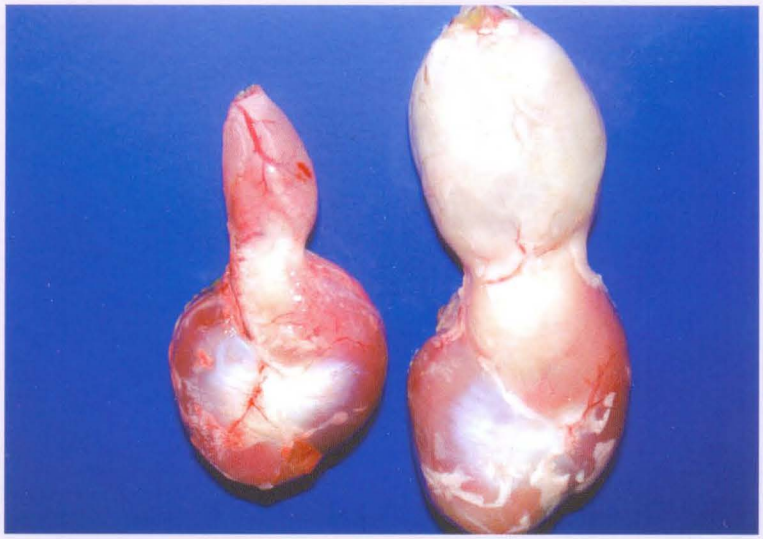


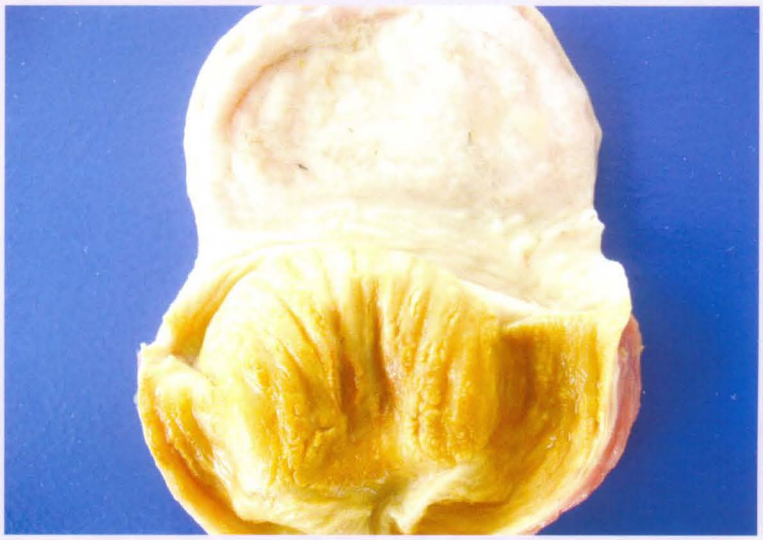
423. Microscopically, the testes of cocks with zearalenone fusariotoxicosis, show a fatty infiltration and atrophy of the germinative epithelium with the exception of the basal layer as well as interruption of the spermatogenesis.

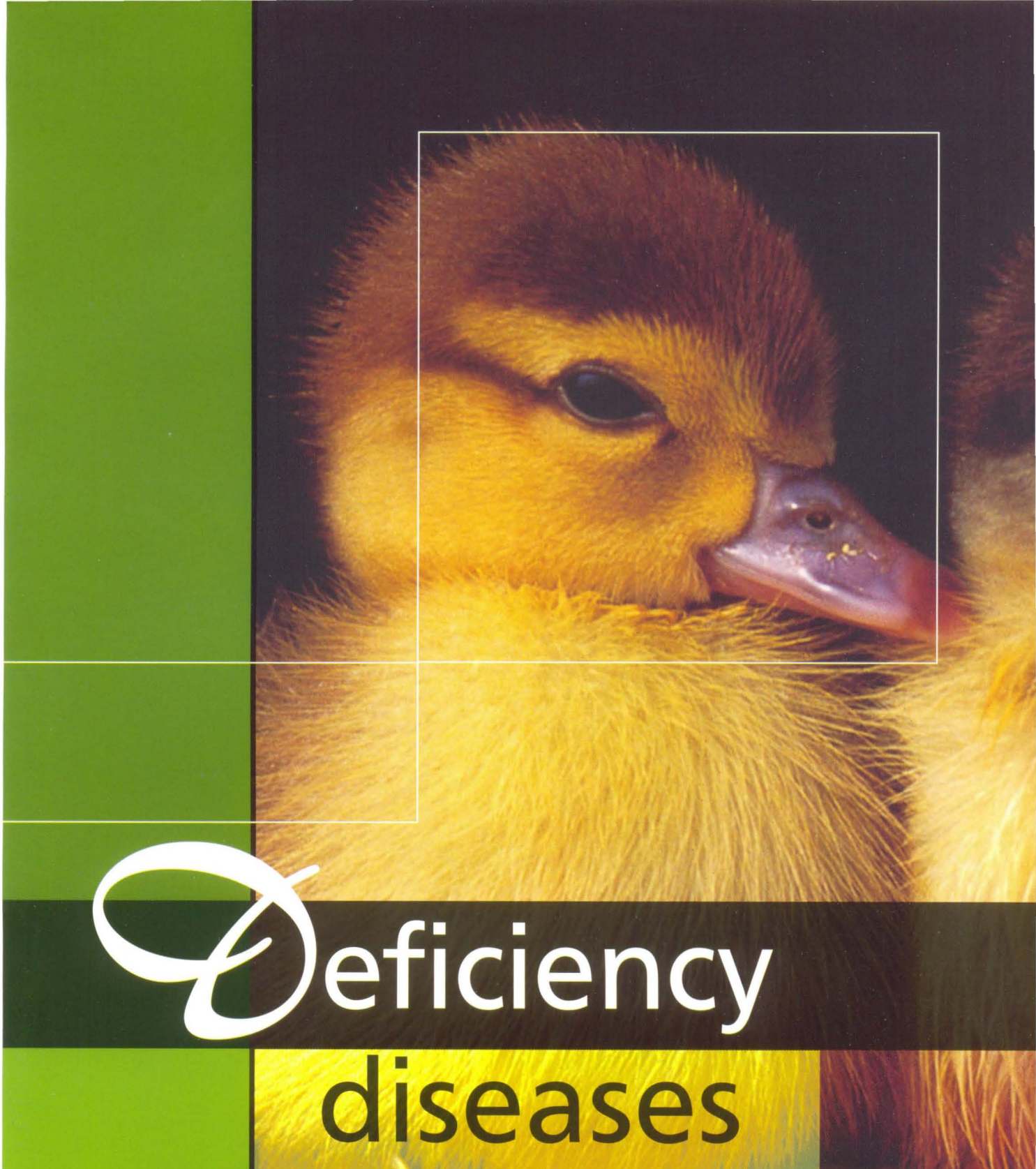


424. Fusarochromanone causes tibial dyschondroplasia in broiler chickens, manifested with long bone deformation.

425, 426, 427.
Frequently, the proventriculus is dilated at a various extent, its wall is thickened and in its mucous coat, fibrinous necrotic and haemorrhagic lesions are detected. The **prevention** from mycotoxicoses requires the detection and control of mycotoxin-contaminated forage components, avoidance of forage moulding and thus, the formation of mycotoxins. A screening of cereals and forages for the presence of some mycotoxins (aflatoxin, T-2 toxin, zearalenone) via ELISA is advised. The application of commercial mycotoxin-binding agents could possibly reduce the effects of some of them. In some mycotoxicoses, the systemic requirements for vitamins, minerals and proteins are increased and they could be balanced by supplementation with forage or water.

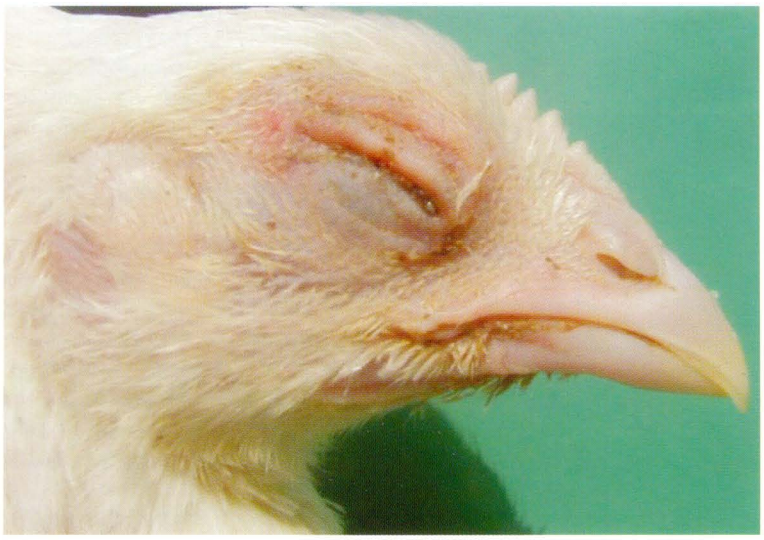






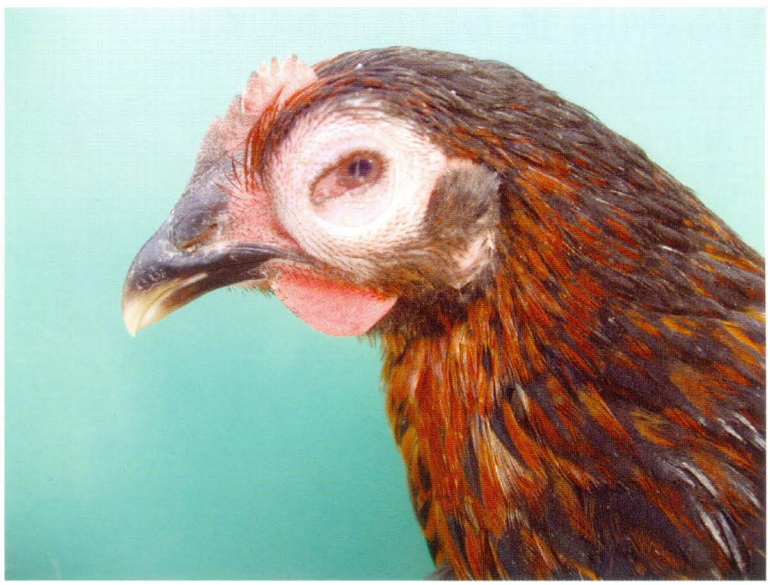
Deficiency diseases

VITAMIN A DEFICIENCY

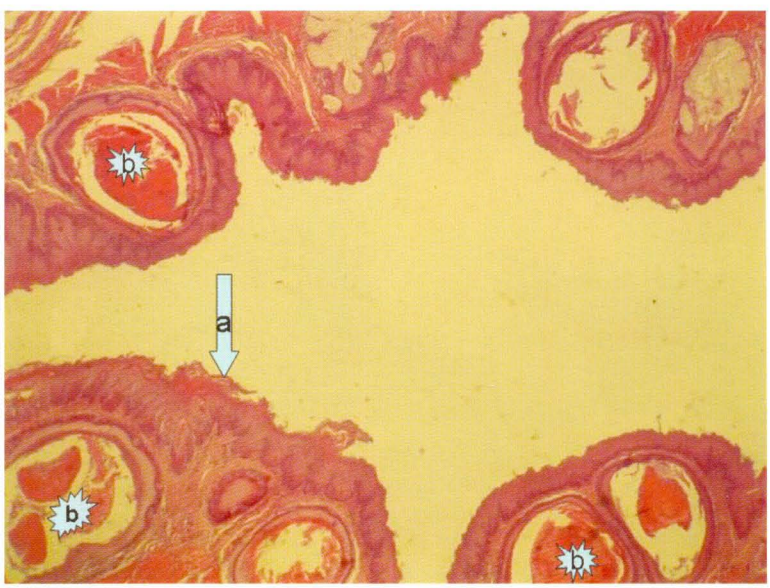


428. In most cases, it is seen in young birds at the age of 1 - 8 weeks. Chickens from the progeny of parents with low vitamin A concentrations, have very low stores and soon after hatching, manifest a deficiency. In recently hatched chickens with vitamin A deficiency, swelling and adhesion of eyelids with a sticky exudate could be observed.

429. In layer hens, dystrophic changes in the epithelia of the conjunctiva and the cornea occur, and later - inflammations following secondary infection, affecting also the adjacent sinuses.



430. A transverse cross-section of oesophagus. In some cases, milliary, grey-whitish, dense prominating nodules could be observed on the surface of buccal mucosa. They are resulting from the hyperkeratinization (arrow **a**) and metaplasia (**b**) of glandular epithelium. The differential diagnosis should take into consideration infectious coryza, chronic fowl cholera, infectious sinusitis in turkeys etc.

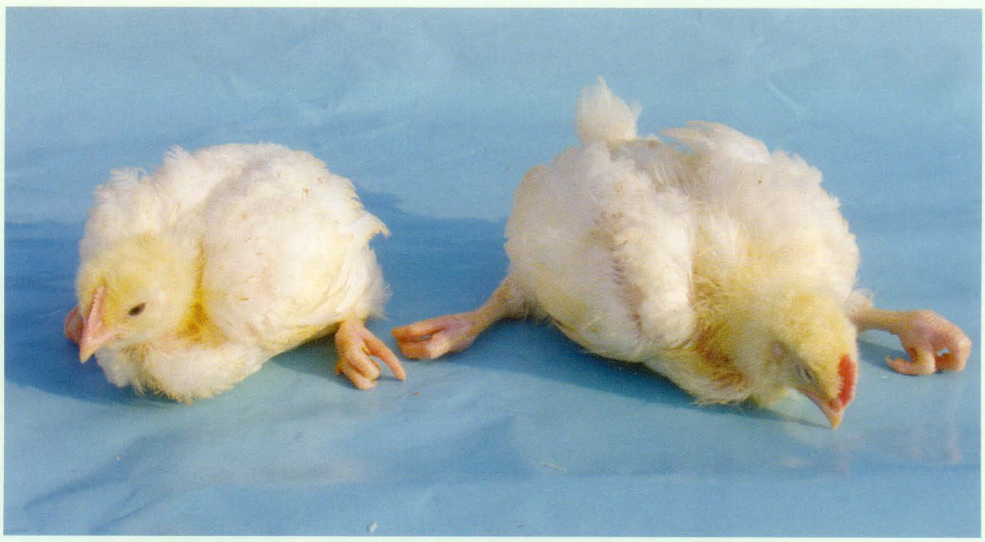


VITAMIN B_1 DEFICIENCY

431. In birds, vitamin B_1 (thiamine) deficiency is clinically and morphologically manifested with paralysis of limbs and muscle atrophy, beginning from the flexors of toes and ascending towards the extensors of legs, wings and the neck. The chickens acquire a specific posture with flexed legs and the head drawn back (stargazing). As hypovitaminosis B_1 causes a severe anorexia, thiamine supplementation to the drinking water is advised until the recovery of acute deficiency and thereafter, the vitamin could be added to the forage.

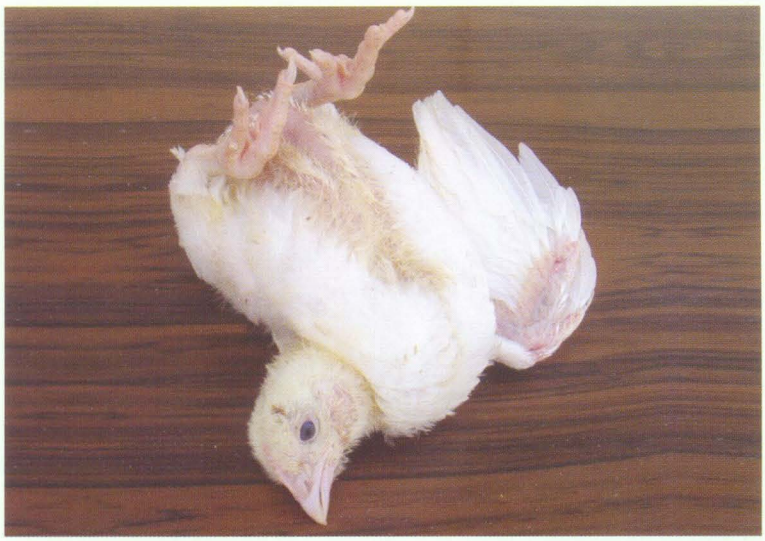
VITAMIN B_2 DEFICIENCY

432, 433. The hypovitaminosis B_2 (riboflavin deficiency) is characterized with impaired oxidation processes and dystrophic changes in the peripheral nerves. The typical clinical sign are curled toes due to paralysis. In the beginning, the toes are slightly flexed and chickens tend to stand on their hocks. In moderate cases, a marked leg weakness and toe flexion in a different extent are observed.



434. In severe cases, the toes are completely curled downward and inward and complete weakness of legs is present. A considerable improvement and relief could be expected if the treatment is initiated in the initial stage of the disease. Water soluble vitamins, that are easily utilized, are recommended.

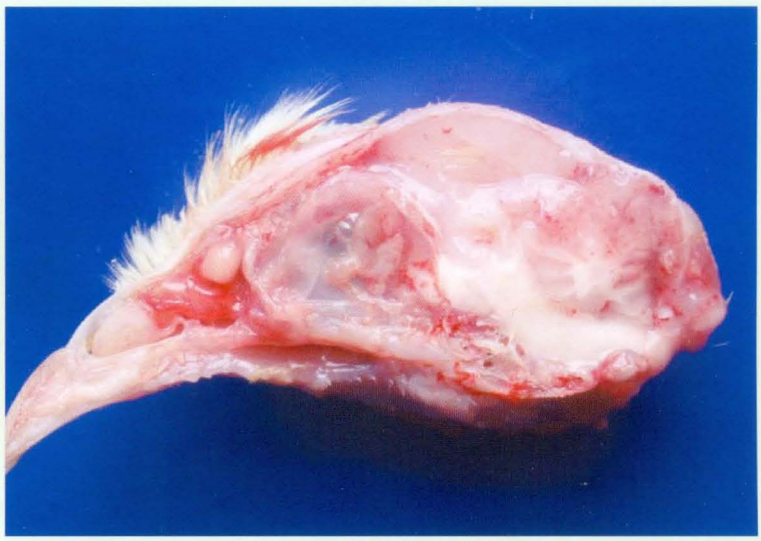
VITAMIN E DEFICIENCY

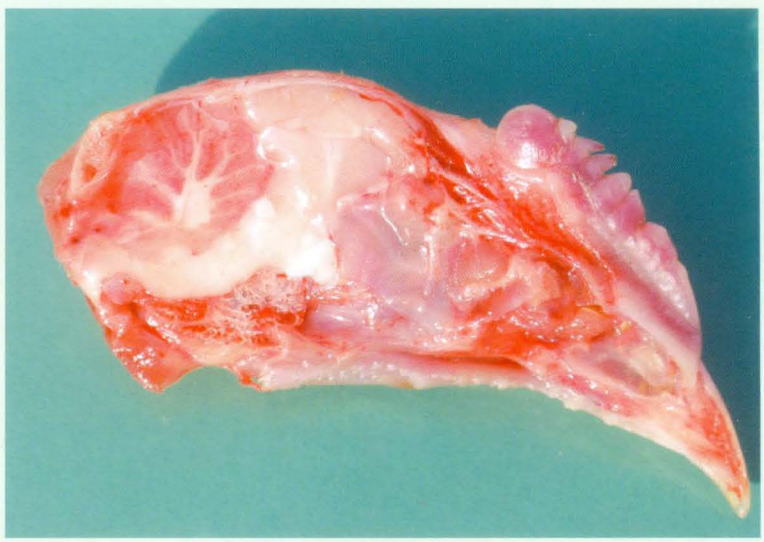


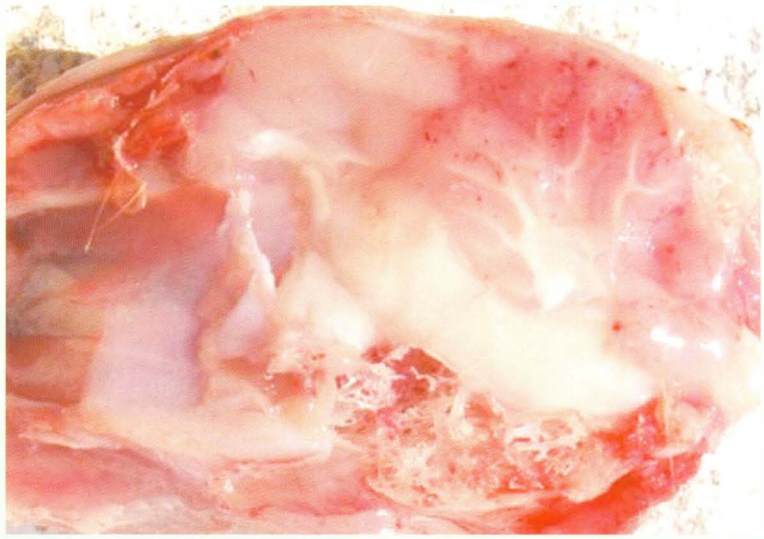
435, 436, 437. The deficiency of vitamin E in poultry is manifested in three different forms: encephalomalacia, muscular dystrophy and exudative diathesis. Each of them is usually encountered independently, although sometimes they occur at a time.

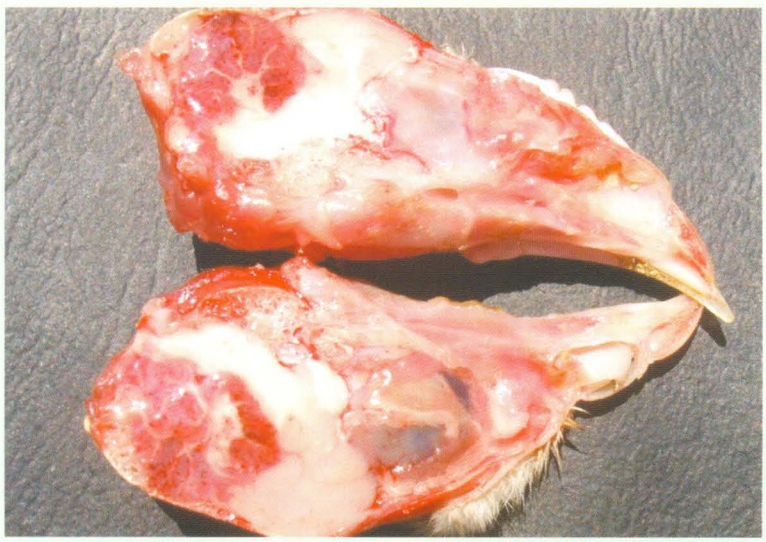
Encephalomalacia (crazy chick disease). The signs are associated to the central nervous system lesions. They include ataxia, disequilibrium, falling on the back, frequent movements with the wings, strongly stretched legs (clonic spasms) and twisting of the head (435 and 436). Rarely, torticolis or opisthotonus (437) could be observed.

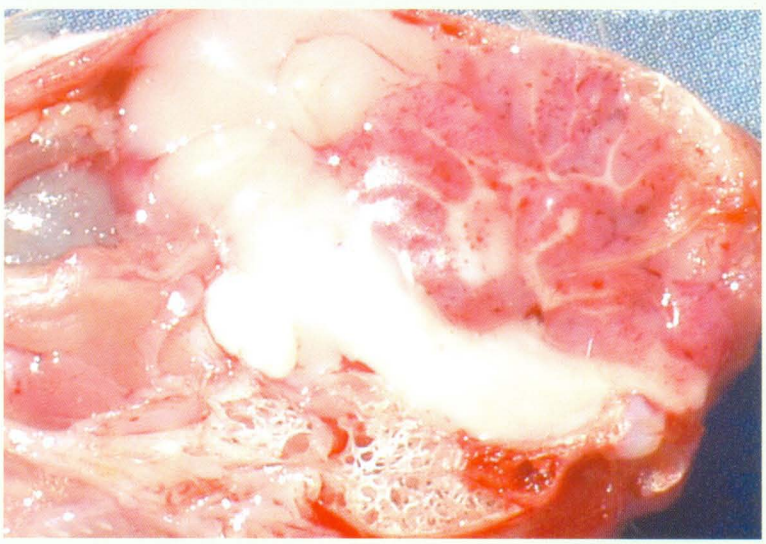
438, 439, 440.
Encephalomalacia is usually observed at the age of 15-30 days but could be also present as early as after the 7th day, as well as after the 56th day of life. Pathoanatomically, oedema, haemorrhages and colliquative necroses are detected in the cerebellum. In the majority of cases, the haemorrhages vary from hardly perceptible to petechial.



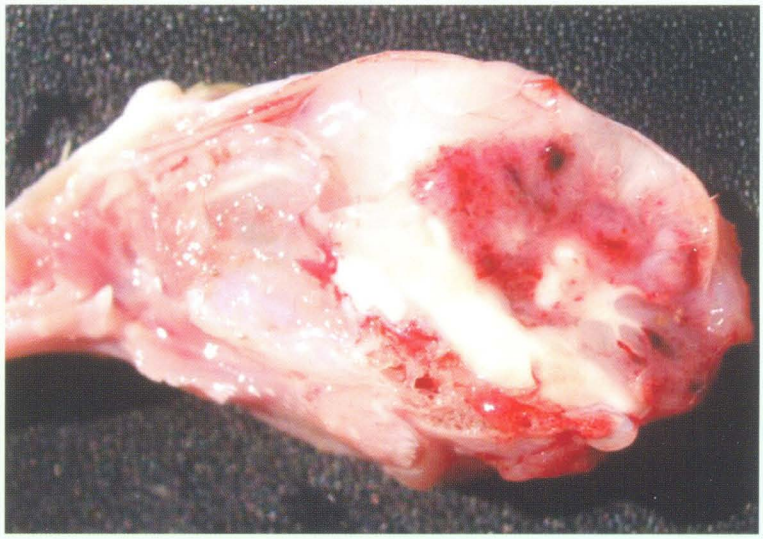


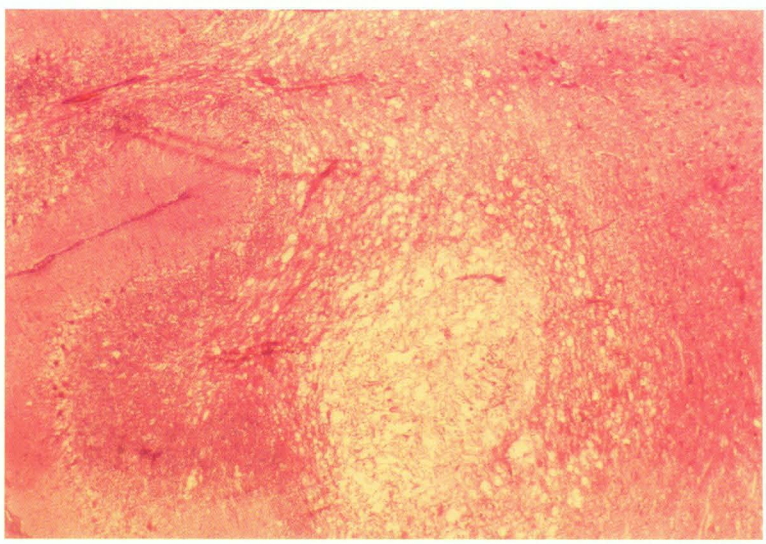


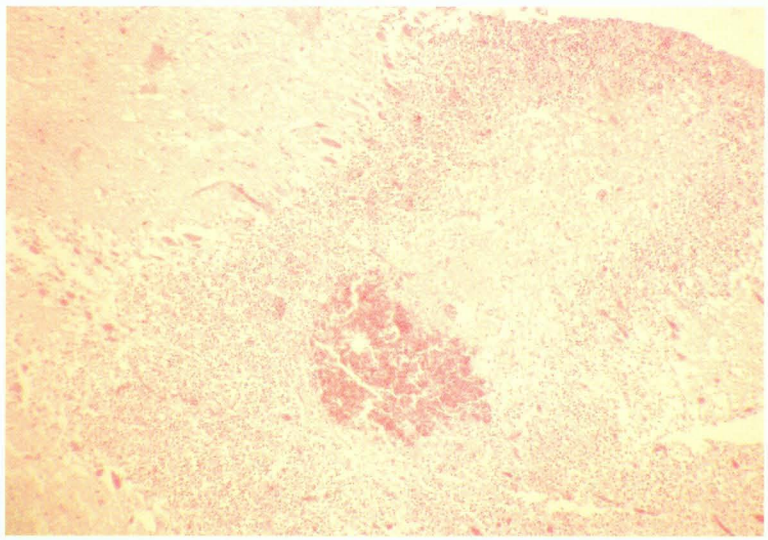




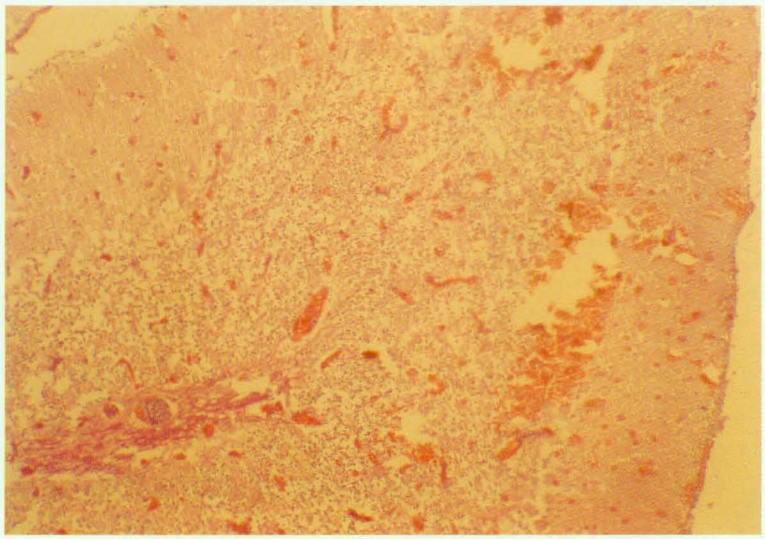
441, 442, 443.
It is possible, although more rarely, to observe massive haemorrhages and sometimes, haematomas in the cerebellum. As an exception, brain lesions could also be present. The vitamin E deficiency is usually manifested in young birds - chickens, turkey poults, ducklings, pheasant poults etc. Most outbreaks are related to high levels of polyunsaturated fat in the diet (meat and bone meal, fish meal etc.) or rancid fat content.



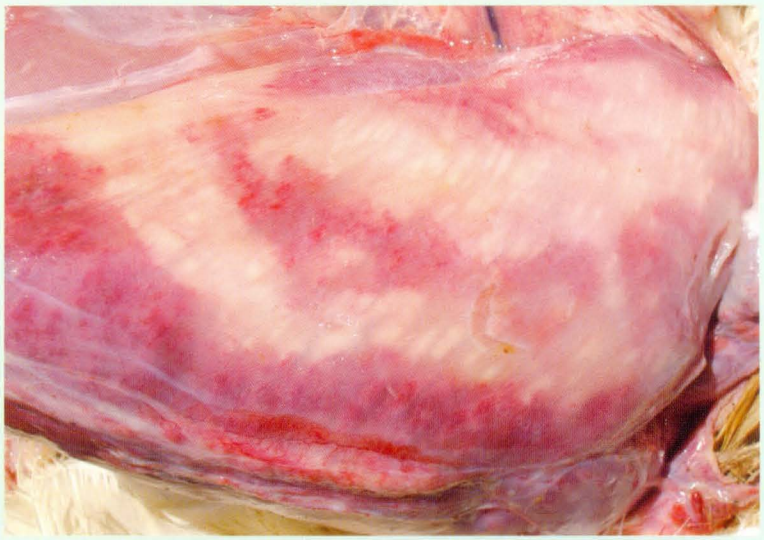


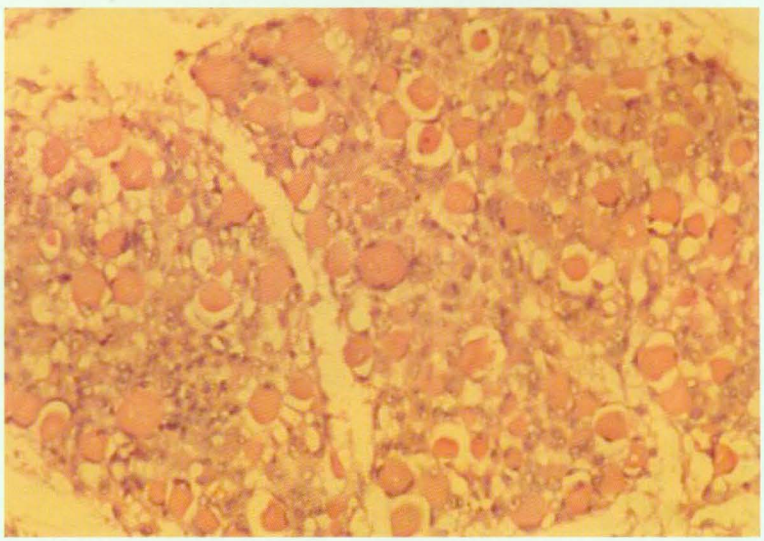


444, 445, 446.
Histologically, colliquative necroses in the white brain substance (444), haemorrhages (445) and multiple thrombosed blood vessels (446) in the cerebellum are detected. Vitamin E and the selenium-containing enzyme glutathione peroxidase preserve the cellular membrane from being destructed by peroxides and other oxidants, produced as metabolic by-products. Peroxides are derivatives of polyunsaturated fatty acids in forages.

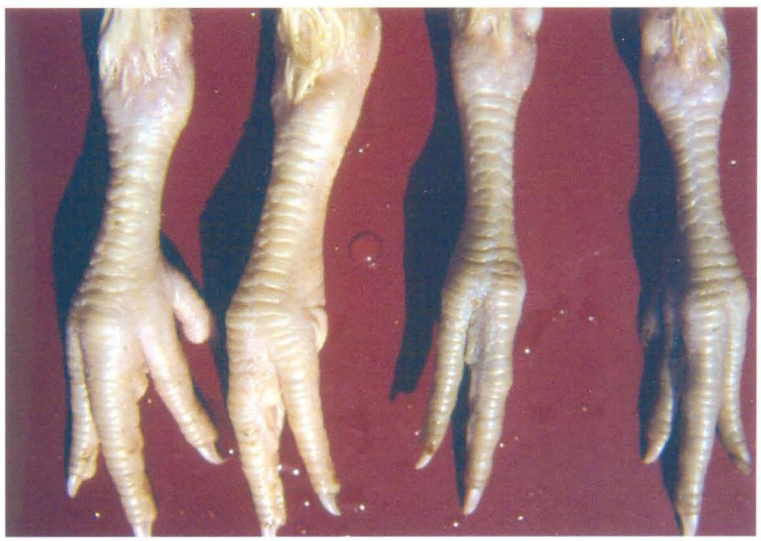


447, 448. Muscular dystrophy. The signs are usually unapparent, but locomotor problems could occur. The pectoral and thigh muscles are most commonly affected, and in them, white or yellowish muscle fibres are found, responsible for the striated appearance of skeletal muscles (447), due to Zenker's necrosis (448).

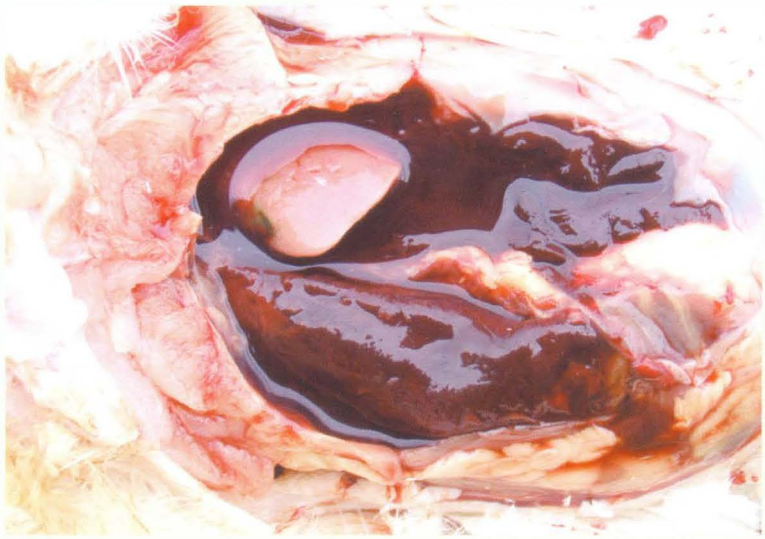


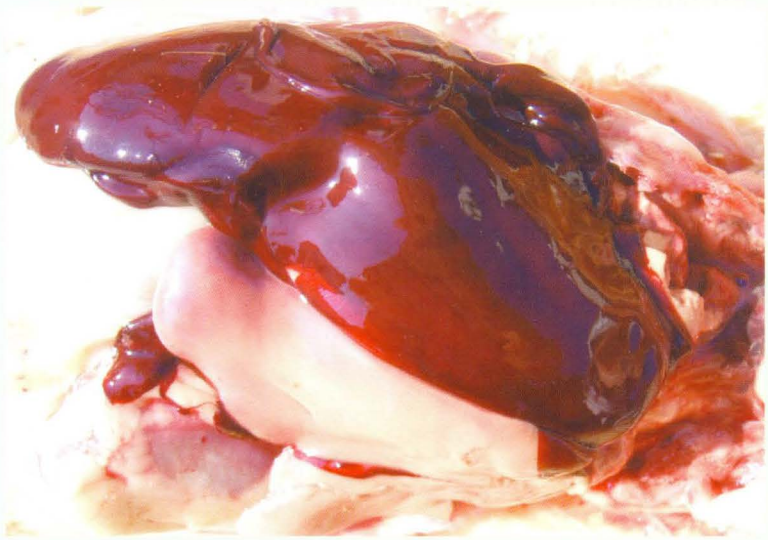


449. Exudative diathesis.
Gelatinous subcutaneous oedemas are discovered in the ventral body parts: breast, abdomen, mandibular space. The skin of legs is often cyanotic. The **diagnosis** is based upon the typical clinical signs and the lesions. The results of the histological examination have a particular value for confirmation of the diagnosis, especially of encephalomalacia and muscular dystrophy. **Control** - utilization of stabilized fats in forages, avoiding the prolonged storage of ready forages for more than 4 weeks. The vitamin E content should be 10 000 IU/tonne and that of selenium - 0.25 ppm.



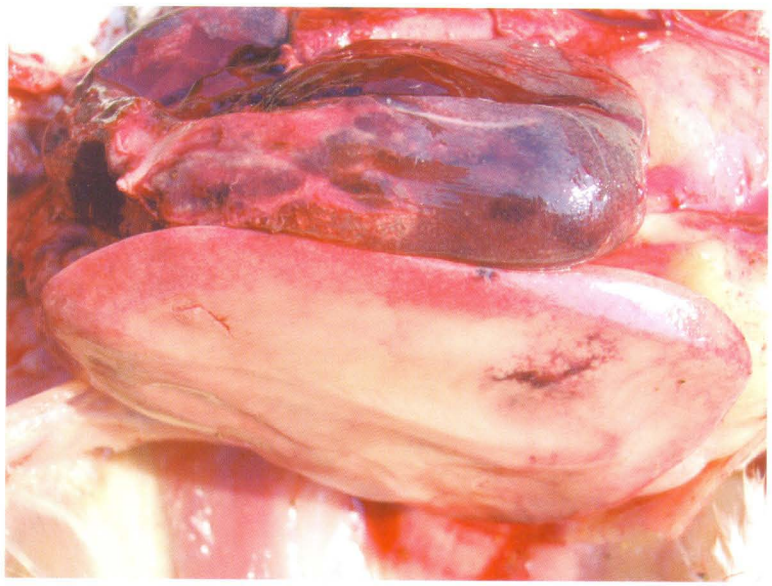
FATTY LIVER HAEMORRHAGIC SYNDROME



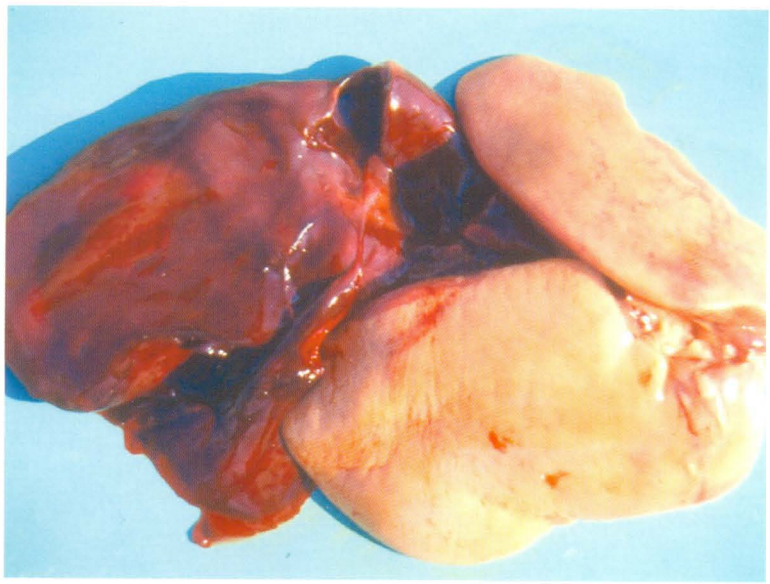


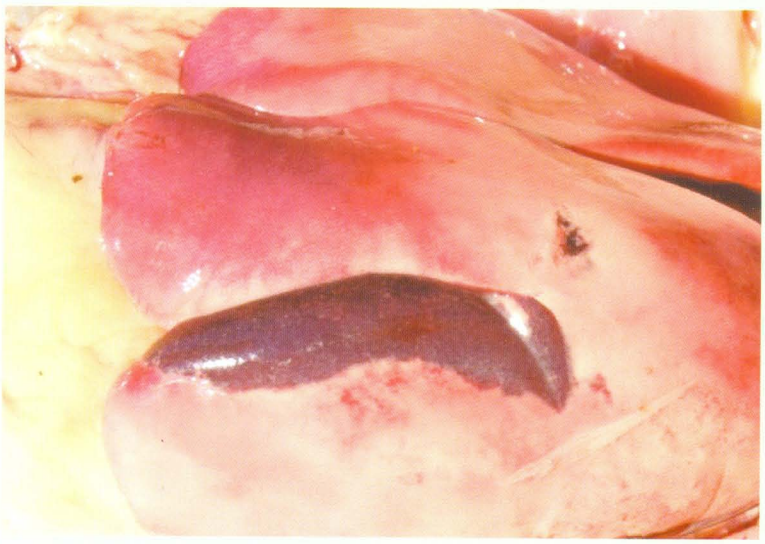
450, 451. The fatty liver haemorrhagic syndrome (FLHS) is a widely prevalent sporadic disease mainly among commercial layers. The FLHS outbreaks are often associated with hot weather and a period of extensive egg-laying. The hens in the flock are overweight (on the average by 20% or more) and a sudden drop in egg production is observed. The birds are discovered suddenly dead, with pale head skin. In the abdomen, large blood clots are detected.

452. The liver is enlarged, pale and brittle.

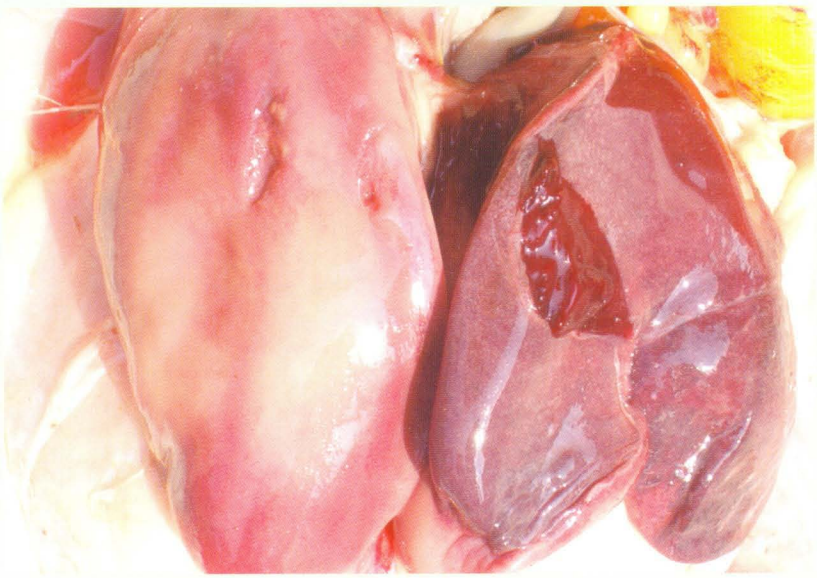


453. In other instances, the organ is yellow, greasy and soft.



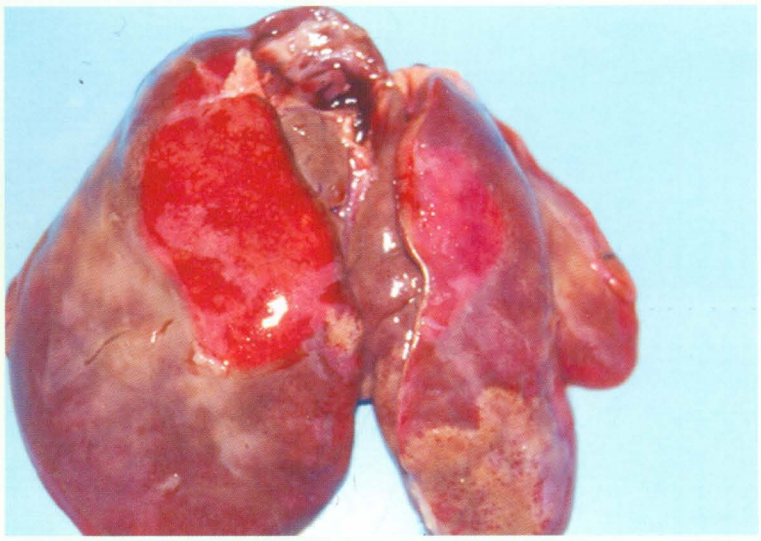


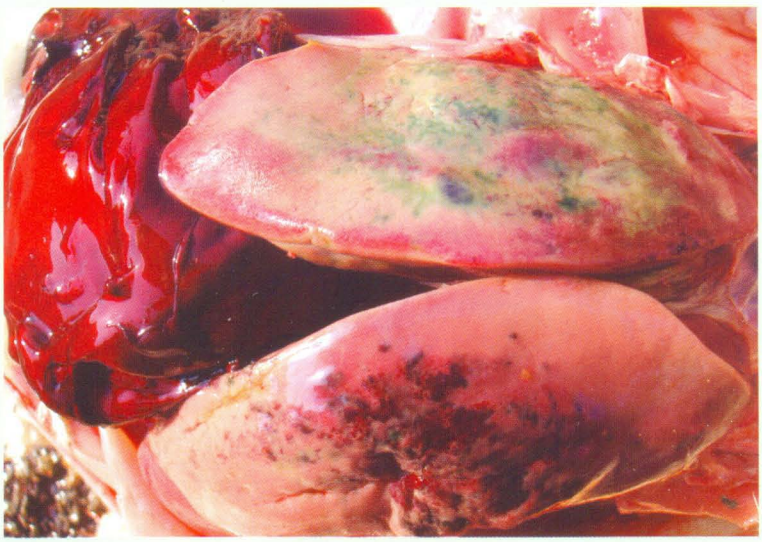
454, 455. Subcapsular parenchymal haematomas are possible. It is assumed that high energy forages and the restricted locomotion are prerequisites for fattening of the liver.



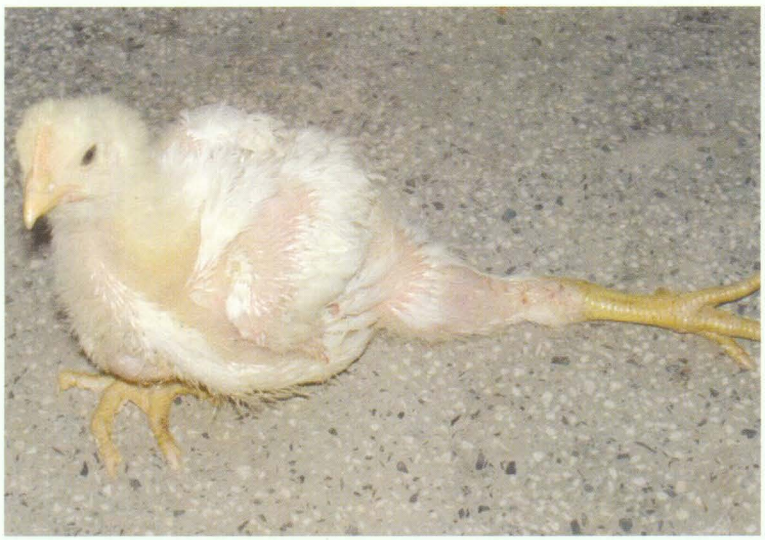
Other possible contributing factors are the deficiency of lipotropic agents, necessary for fat mobilization by the liver, aflatoxins, genetic factors etc. Frequently, FLHS and cage layer fatigue are diagnosed at a time.

456, 457. Clinically healthy birds in the flock could also exhibit liver haematomas, dark red (fresh) or green to brown (old). Considerable amounts of fat are detected in the abdominal cavity. The only successful approach for prevention is the reduction of obesity in layers. The use of lipotropic agents such as vitamin E, vitamin B_{12} and choline chloride gives conflicting results. The avoidance of heat stress and moulded forages could be also helpful.



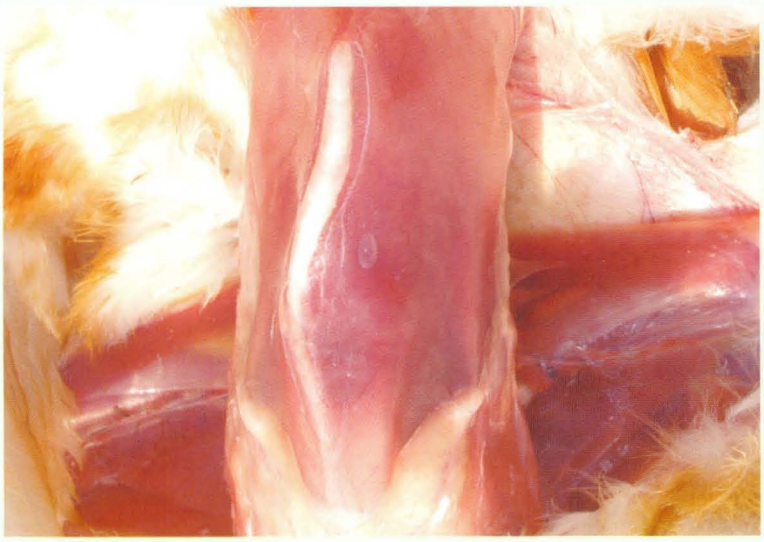


SLIPPED TENDON (PEROSIS)



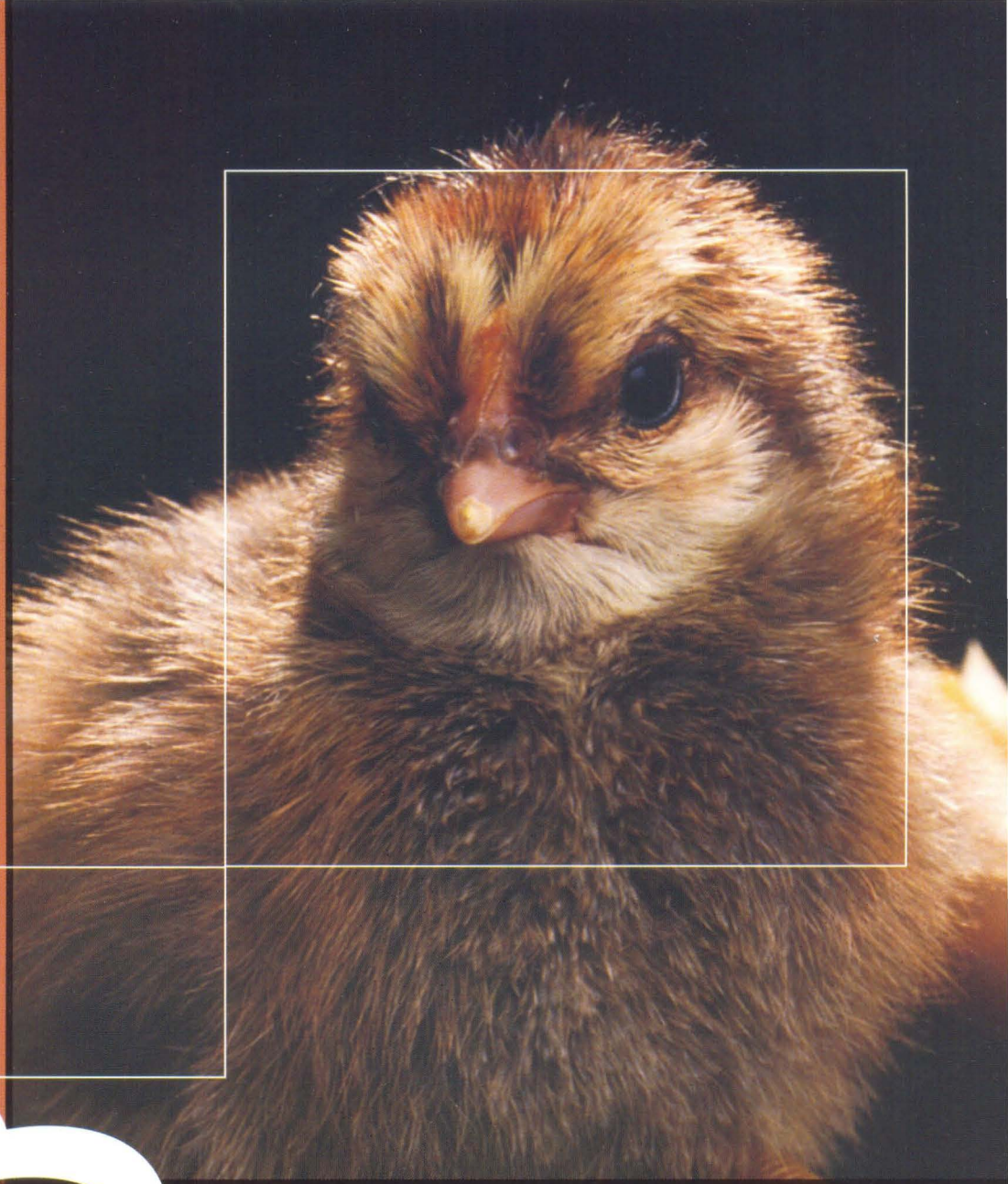
458, 459, 460.
Perosis or chondrodystrophy is encountered in young birds whose diet is deficient in manganese (Mn) or some of the following vitamins: choline, nicotic acid, pyridoxine, biotin or folic acid. This is an anatomic deformation of leg bones in young chickens, turkey poults, pheasant poults etc. It is characterized by retarded growth of long bones, widening of the tibiometatarsal joint, twisting or bending of the distal end of tibia and the proximal end of metatarsus and finally, slipping of the gastrocnemius muscle tendon from its condyles. Clinically, it is manifested by impaired locomotion because of leg lateral and posterior malposition of the leg.

RACHITIS



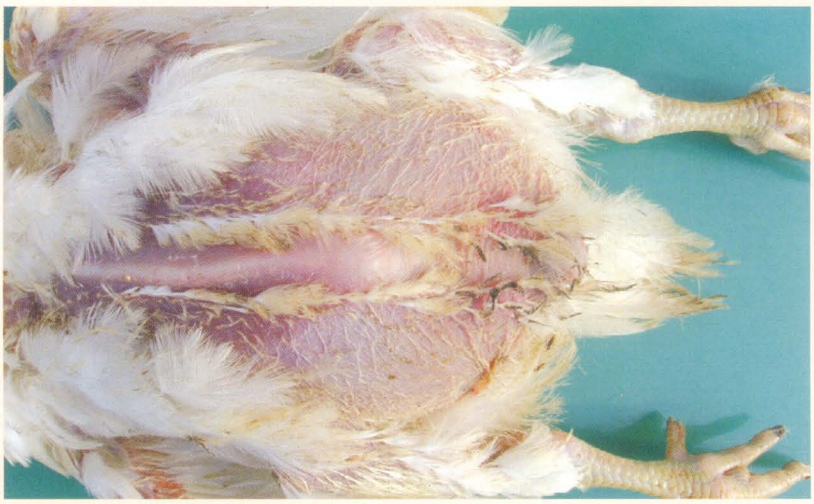
461, 462. The vitamin D_3 deficiency or lack, or the impaired ratio of calcium and phosphorus result in rachitis in young birds. It is encountered more frequently in industrial poultry breeding where the forages are often inadequately balanced. The deficiency of vitamin D_3 and phosphorus are more common in growing birds whereas the calcium deficiency in young or adult layer hens. The newly hatched chickens have depleted calcium stores. The calcium deficiency is rapidly manifested if they do not receive an adequate diet supplement. Clinically and morphologically, soft bones or various degree of bone deformation are determined. The birds usually lie down and their growth is retarded. The **diagnosis** is based upon the complex evaluation of their age, signs and lesions.

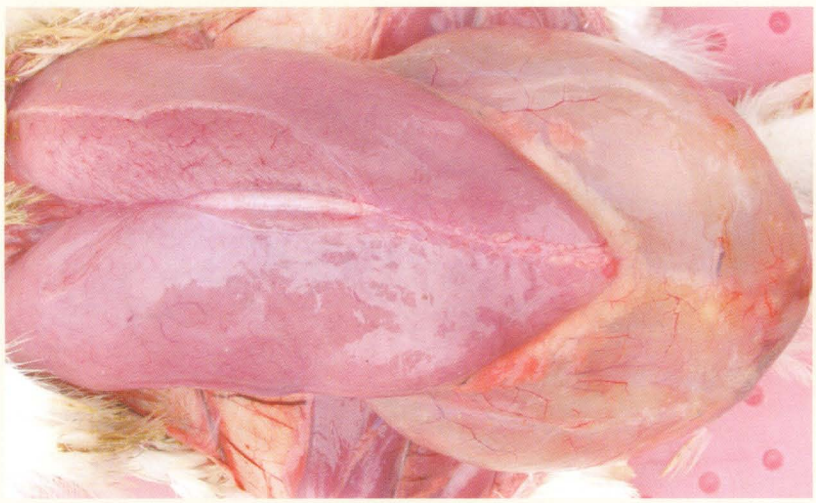
The supplementation of vitamin D_3 with water or forage and the balancing of Ca/P ratio in the diet contribute to the favourable outcome of the disease.



Others

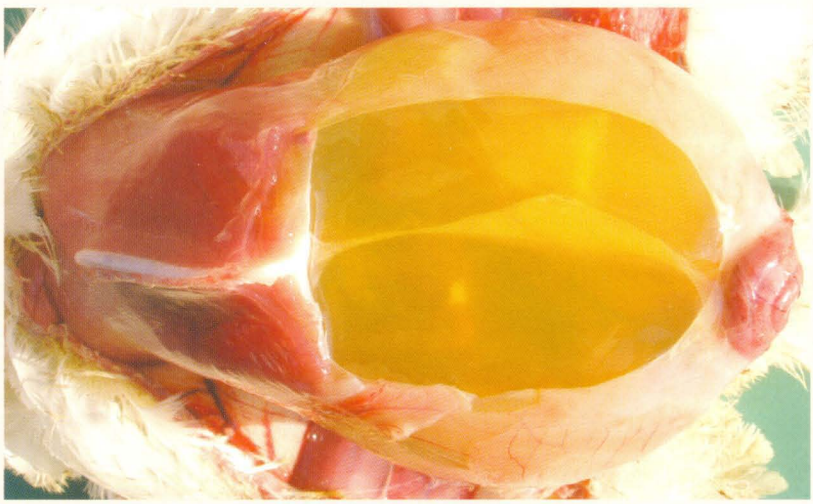
PULMONARY HYPERTENSION (ASCITIS) SYNDROME IN BROILER CHICKENS

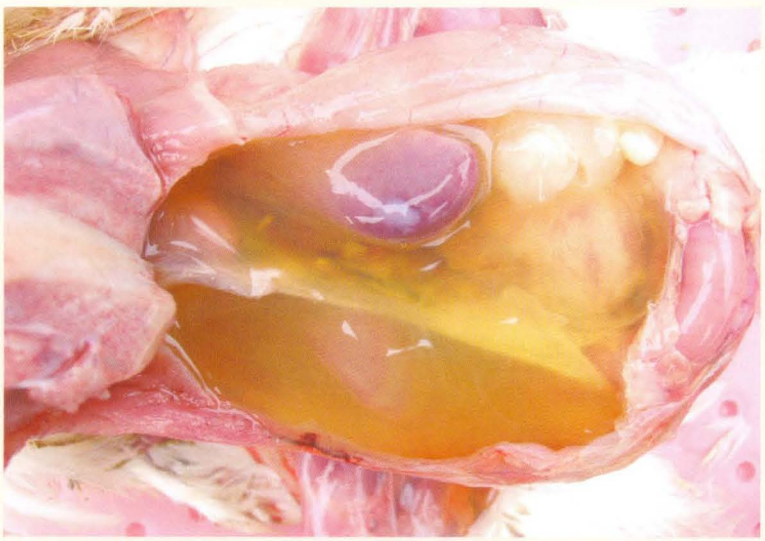




463, 464. The pulmonary hypertension syndrome (PHS) or ascitis is related to the rapid growth and enhanced metabolic processes in broilers. Affected chickens are with a strongly distended abdomen, reluctance to move, respiratory troubles and cyanosis.

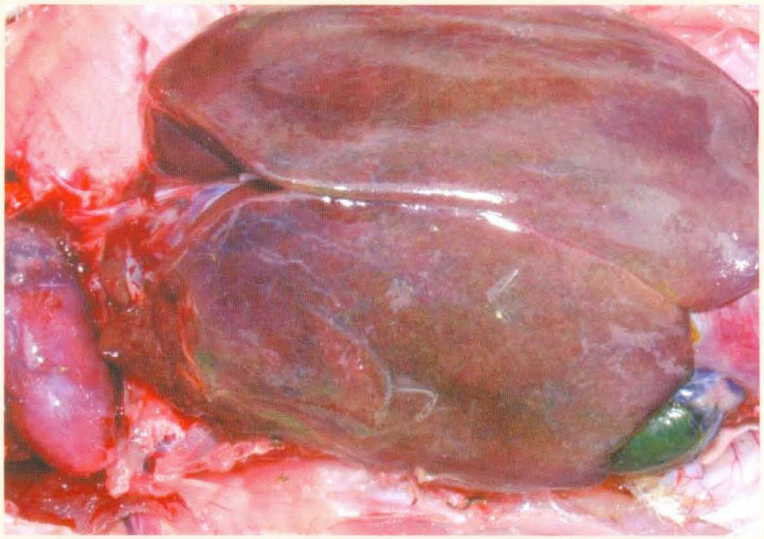
465, 466.
The pleuroperitoneal cavity of affected chickens is filled with straw-yellow fluid. The rapid growth in contemporary broilers is related to higher needs for oxygen, and the lung remains relatively small vs body dimensions.

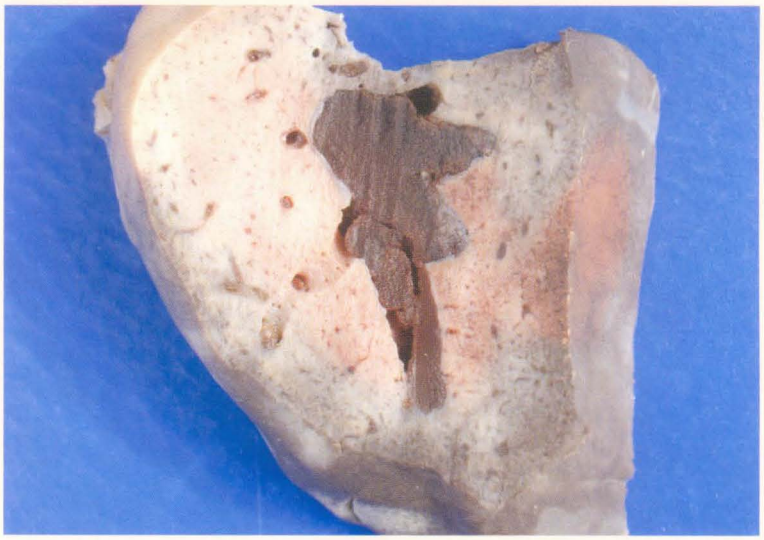




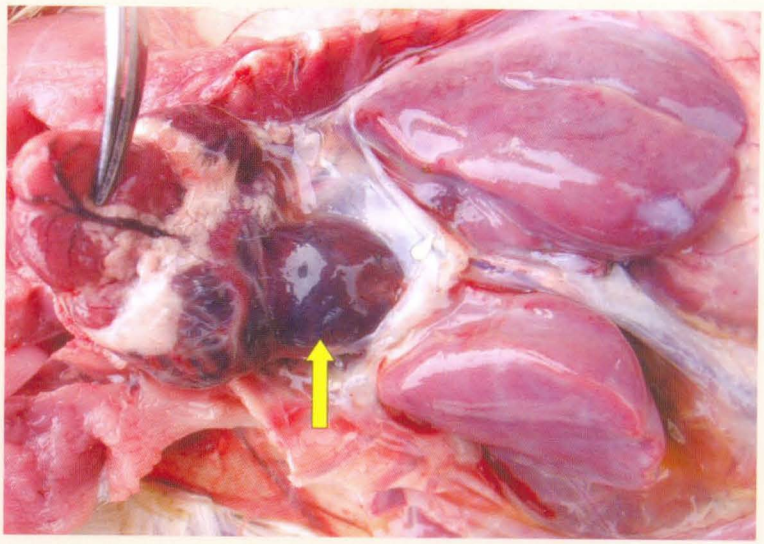
The main factor for PHS is hypoxaemia that results in enhanced cardiac activity. The consequences are pulmonary hypertension, right cardiac failure and ascitis.

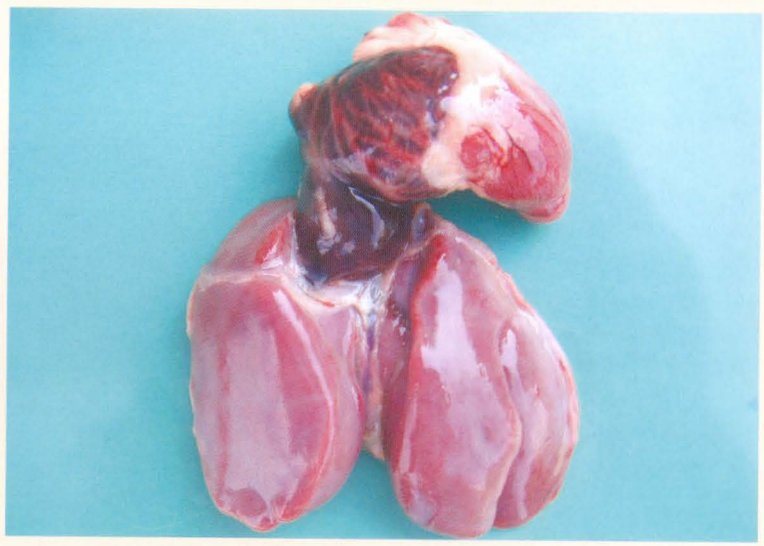
467, 468. Immediate causes for hypoxaemia are the inadequate ventilation, the high ammonia or dust levels in premises, low temperatures, stimulating the metabolic processes. Generalized passive venous hyperaemia and stasis in the liver and other viscera are observed.

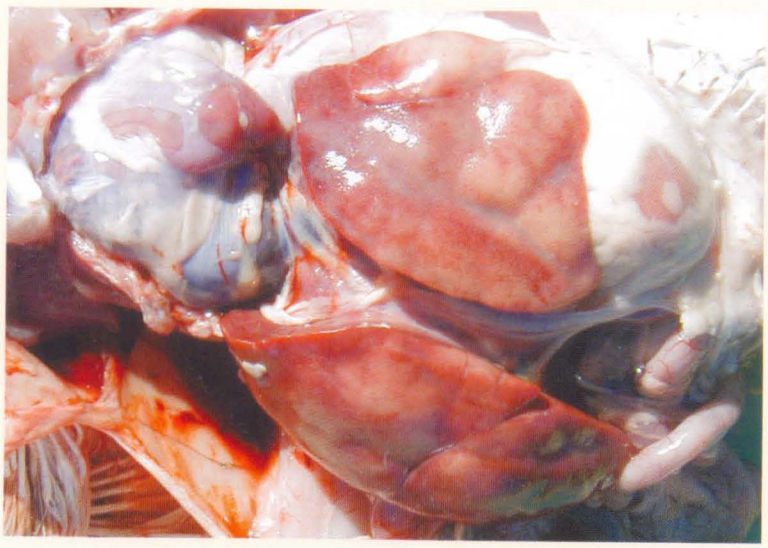




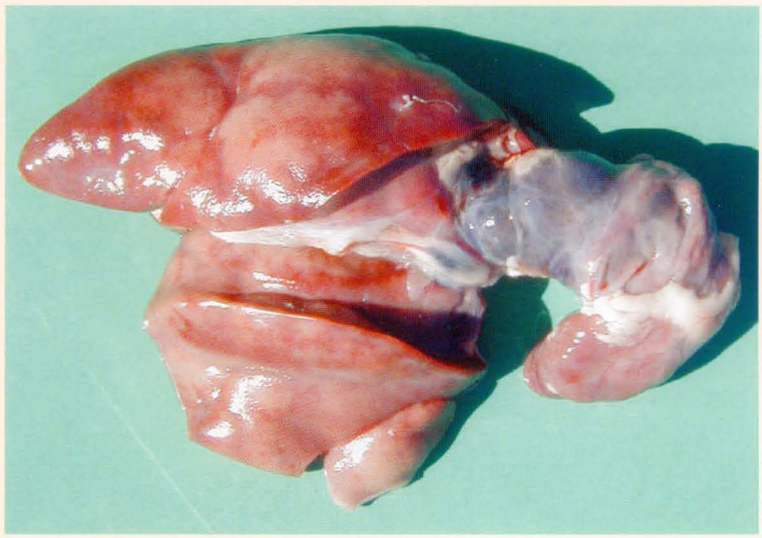
469, 470. The posterior vena cava and the right half of the heart are overfilled with blood.

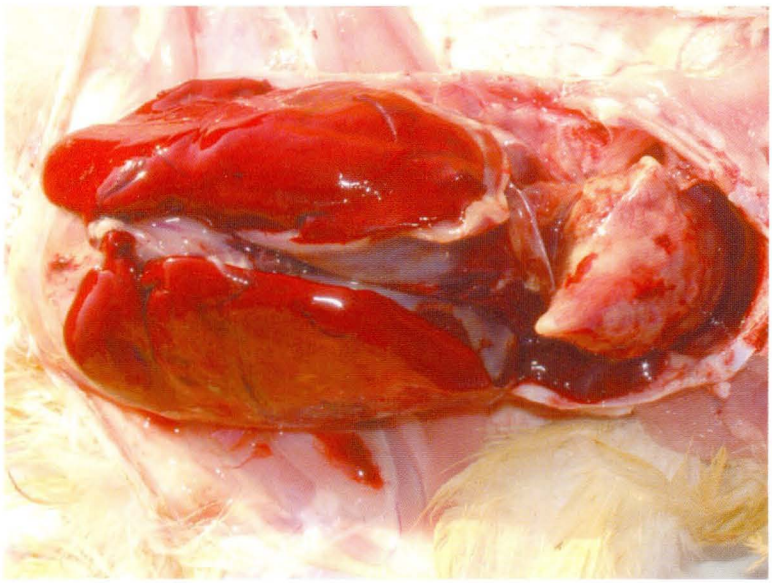






471, 472. In advanced stages, regenerative reparative processes in the liver could occur. The hypoxia favours the growth of connective tissue in the interstitium that results in thickening of the organ and appearance of sclerotic lesions.

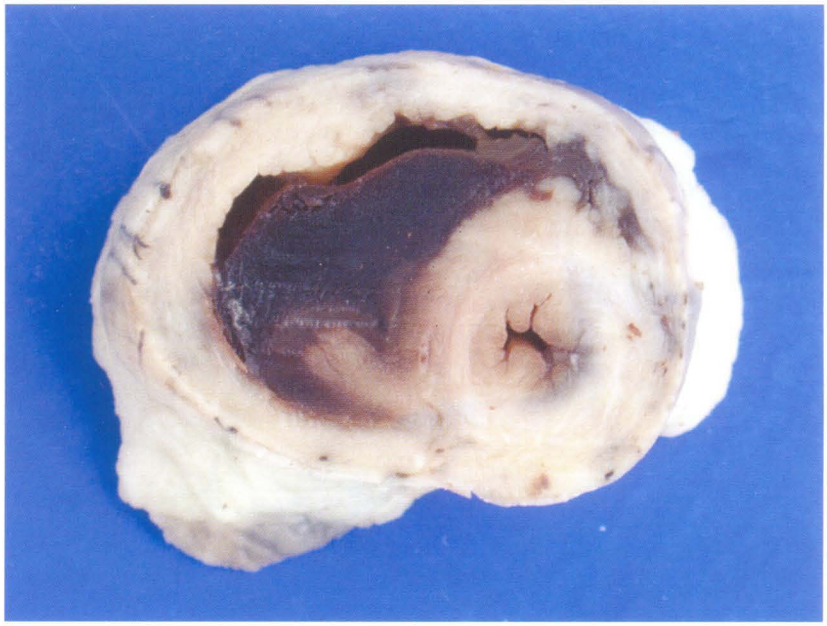


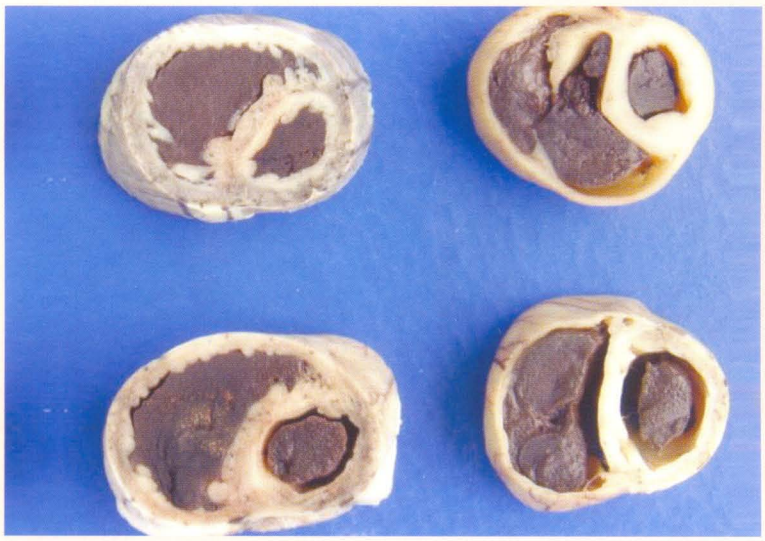


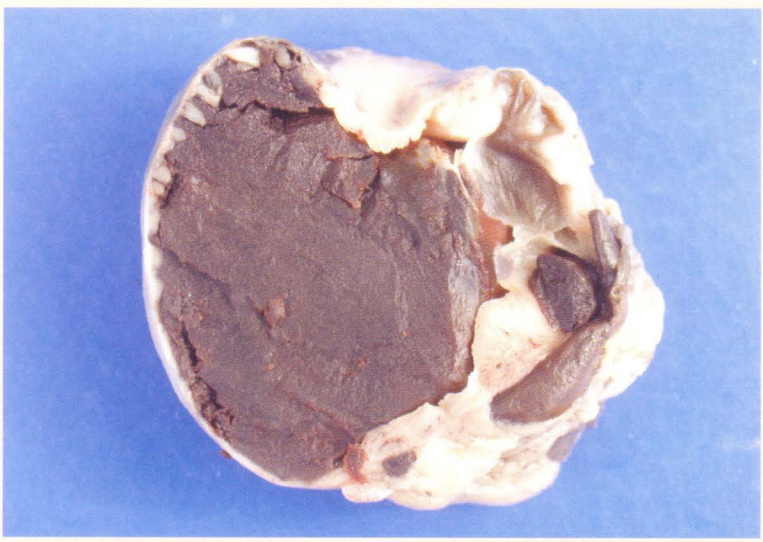
473. The stasis is sometimes causing liver rupture and internal bleeding.

474. In a number of cases, a secondary contamination with *E. coli* could occur and therefore, a serous fibrinous inflammation, manifested as poly-serositis, could be observed.

475. Often, a left compensatory hypertrophy is observed (transverse cross-section of a fixed heart).

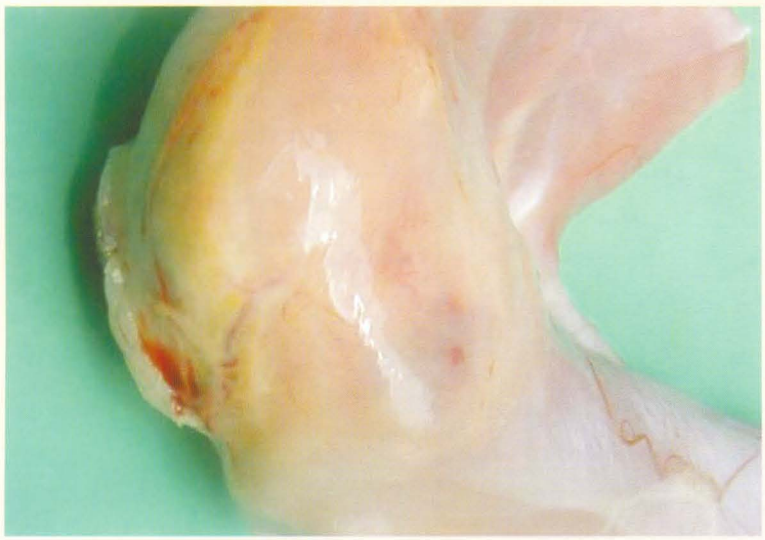


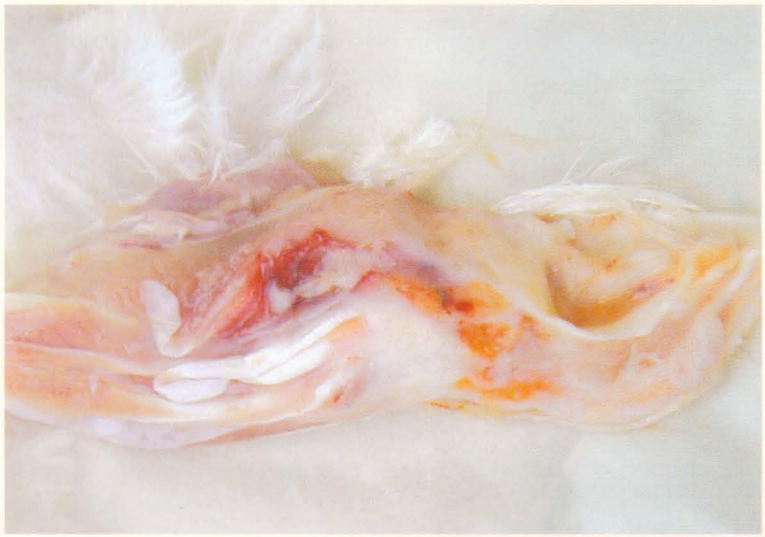




476, 477. The right ventricle at an early stage is hypertrophied whereas at a later - dilated with extremely thinned wall (transverse cross sections of fixed heart). The **diagnosis** is based on gross lesions. The reduction of oxygen requirements by slowing down the metabolism protects from and decreases the incidence of ascitis. Restriction of forage consumption and various light patterns are recommended.

AMYLOIDOSIS



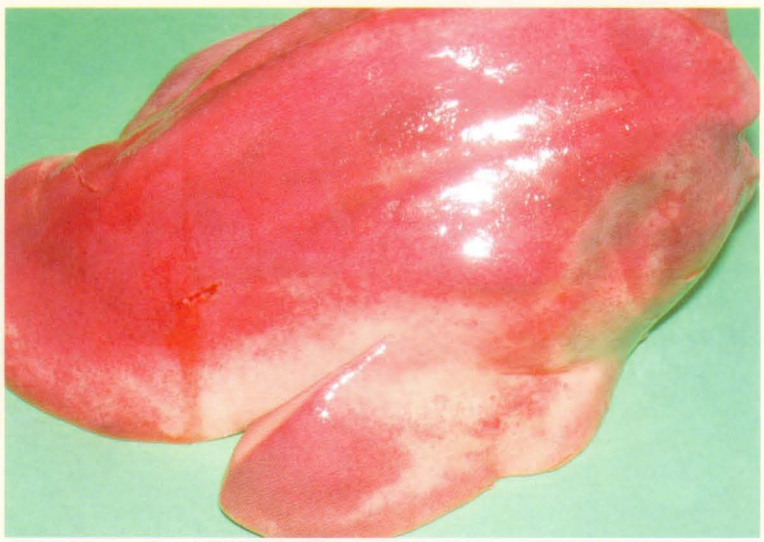


478, 479. Amyloidosis is seen mainly in adult birds. It is characterized by extracellular buildup of the protein amyloid in different viscera and the joints.

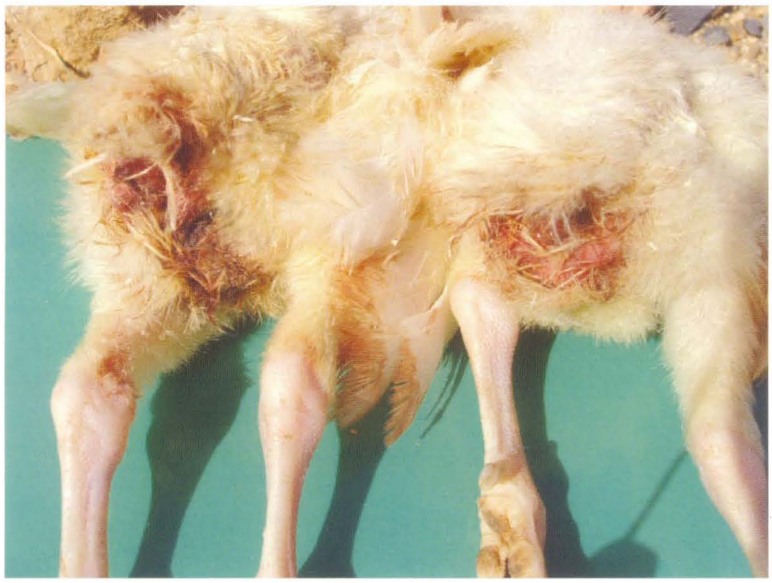
Amyloid arthropathy.

It is primarily associated with *Enterococcus faecalis* and *Mycoplasma synoviae*. Brown layers are particularly susceptible. Some genetic factors could be involved in the incidence of amyloidosis too. The affected joints are enlarged, swollen and contain orange-yellowish matter.

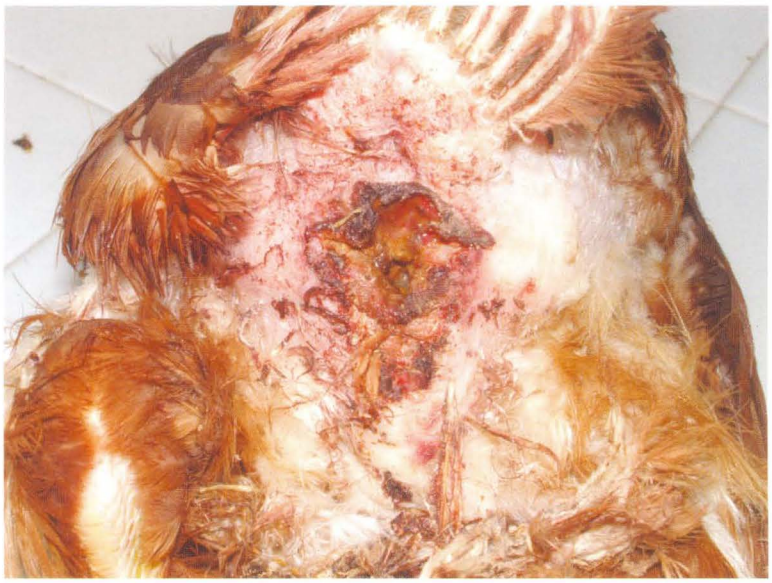
480. Amyloidosis of internal organs. The buildup is observed mainly in the liver, the spleen and the kidneys. The affected organ is multifold enlarged, with stretched capsule, rounded margins and pale colour. The state is usually seen following severe disturbances in protein metabolisms prolonged and exhausting diseases (tuberculosis etc.).



CANNIBALISM

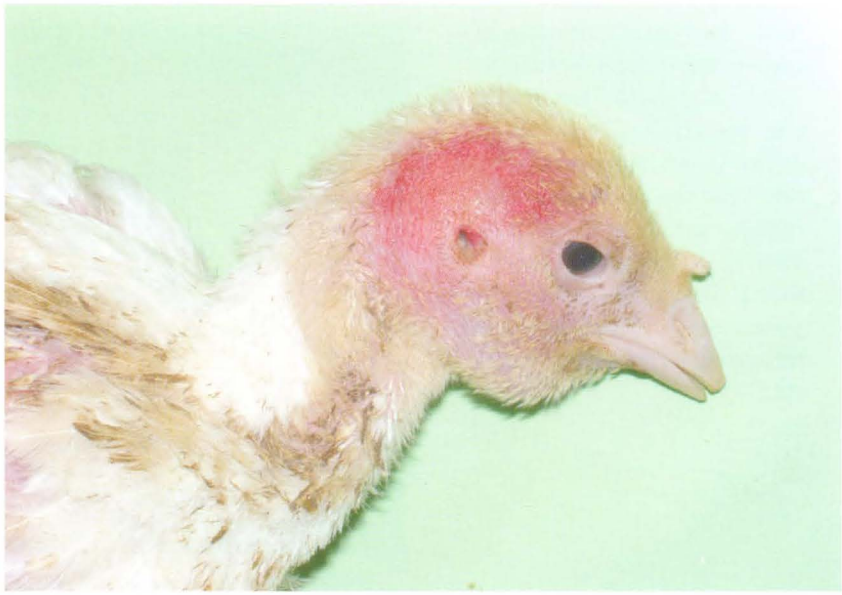


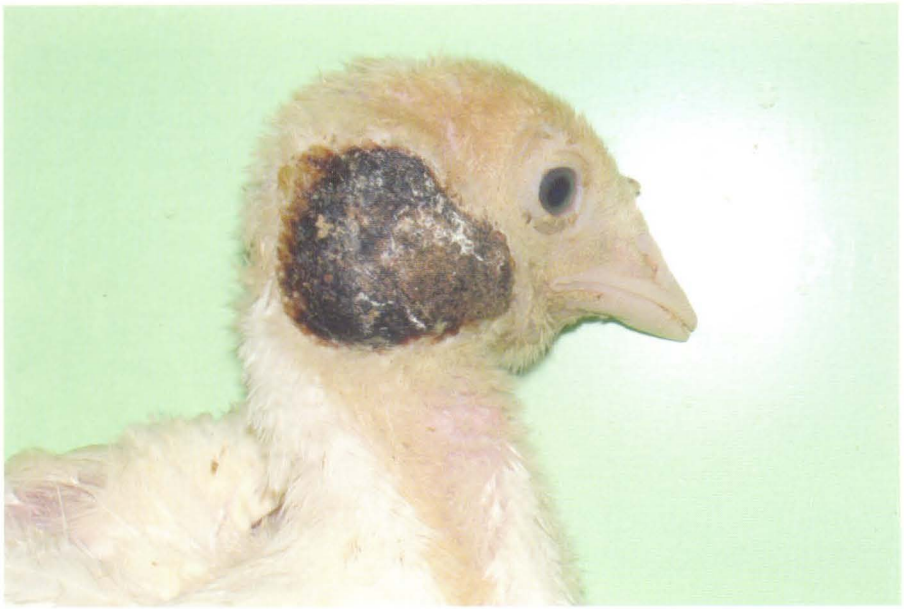
481. The cannibalism in birds is usually consequence of feather pecking or pulling out, or vent pecking, that are behavioural reactions. Feather pecking or pulling out are observed in birds, reared in closed, often overcrowded premises. This happens frequently in growing broiler breeder flocks during the period of restricted feeding.



482. The vent pecking is usually taking place soon after the beginning of egg-laying and could be related to occurring hormonal alterations.

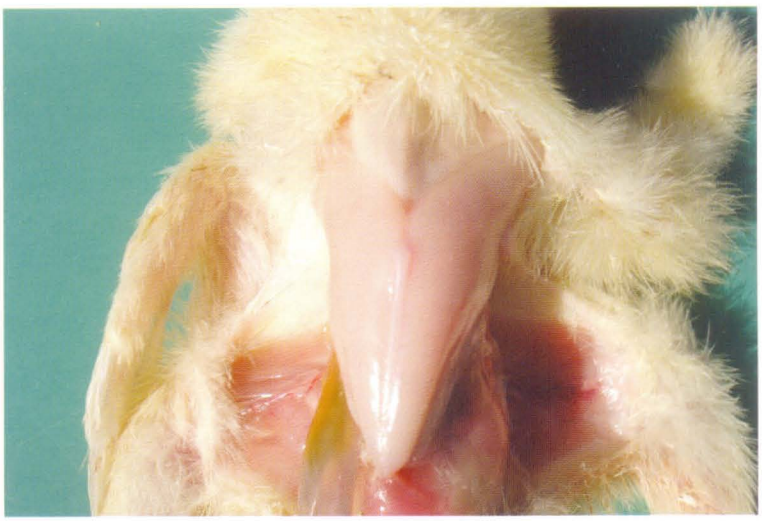
483. The pecking of the skin in various parts of the body and the vent result in blood loss, protrusion of viscera from body cavities and death. Some of the states that are supposed to provoke feather pecking are the high light intensity in the premise, pelleted feed, nutritional and mineral deficiencies and skin injury by ectoparasites.





484. Formation of crusts after pecking in the head area in a turkey poult. The prevention of cannibalism could be done by providing adequate diets (ground feeds are preferred), reduced light intensity, avoiding overcrowding. An extreme measure is debeaking.

485. Marked anaemic appearance of the carcass due to massive blood loss subsequent to cannibalism in a turkey poult.

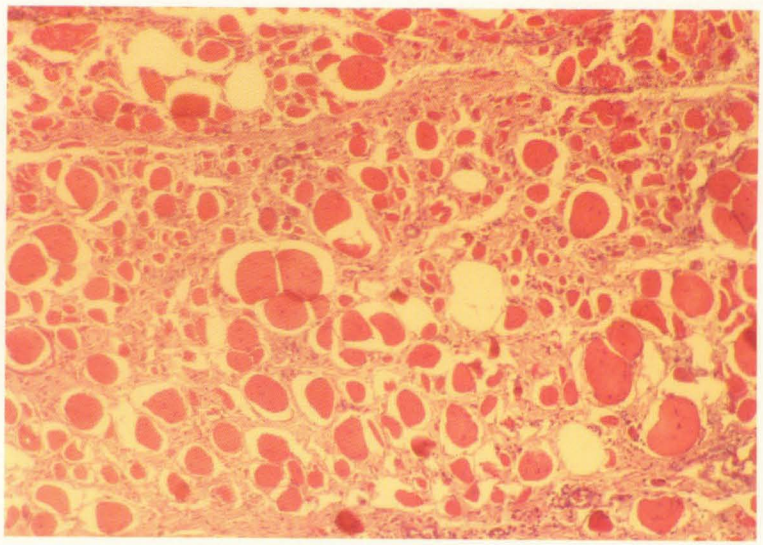


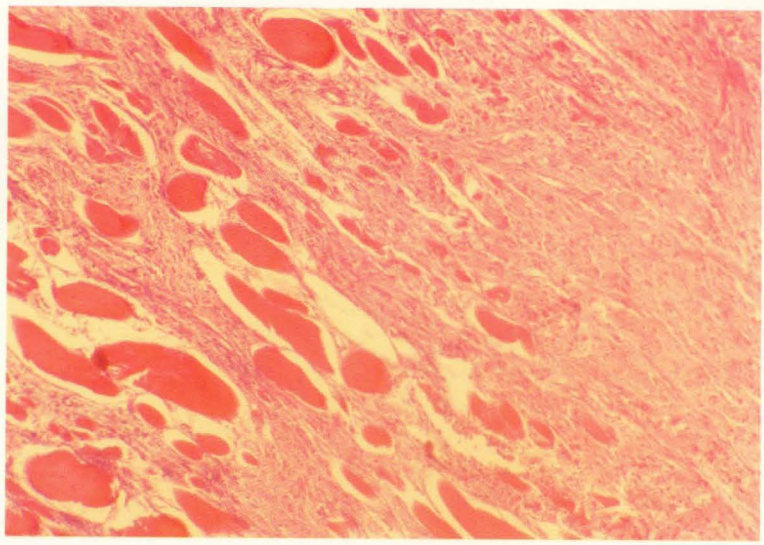
DEEP PECTORAL MYOPATHY

486, 487. The deep pectoral myopathy (DPM), called also green muscle disease or Oregon disease, is observed in heavy meat types of turkeys or chickens. The disease occurs because of ischaemic necrosis due to inadequate blood supply of variously sized deep pectoral muscle groups.

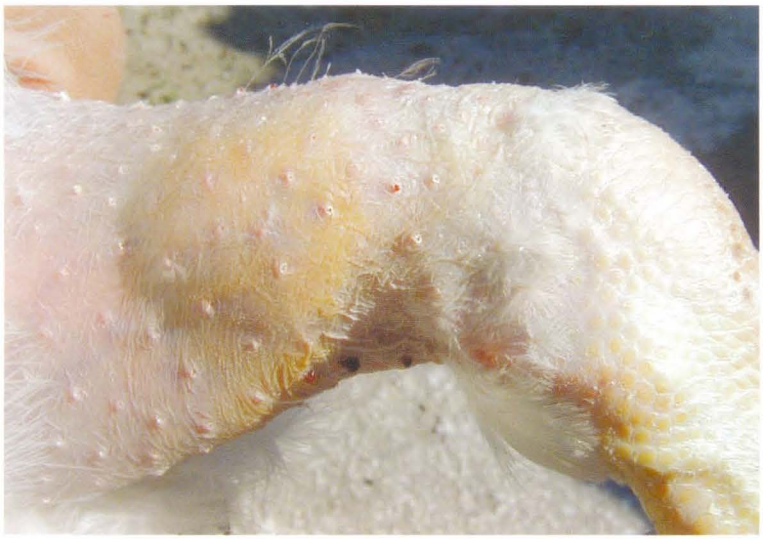
The lesion is uni- or bilateral and is detected as a slaughterhouse finding. Affected muscles have an unusual green colour.

488, 489. Histologically, the altered muscle fibres are enlarged at a various extent, markedly eosinophilic with rectic or lacking nuclei. At a more advanced stage, among the necrotized tissue, inflammatory reaction and replacement of atrophied fibres with fat or fibrous tissue could be seen.

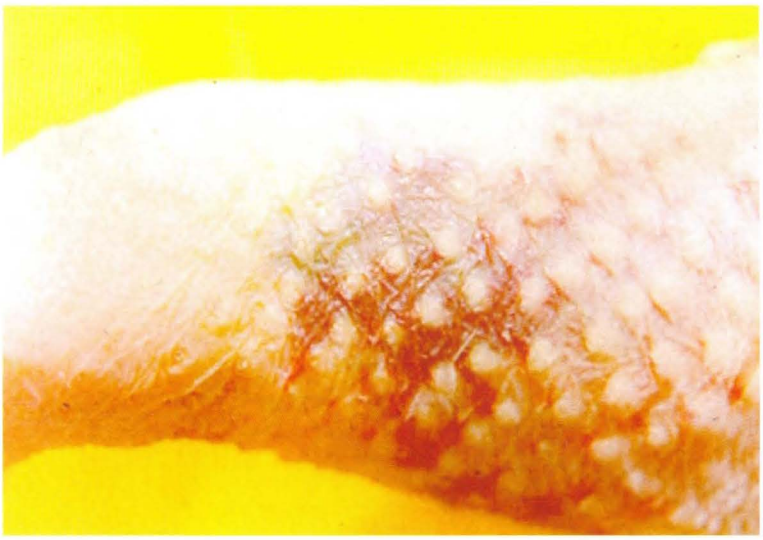




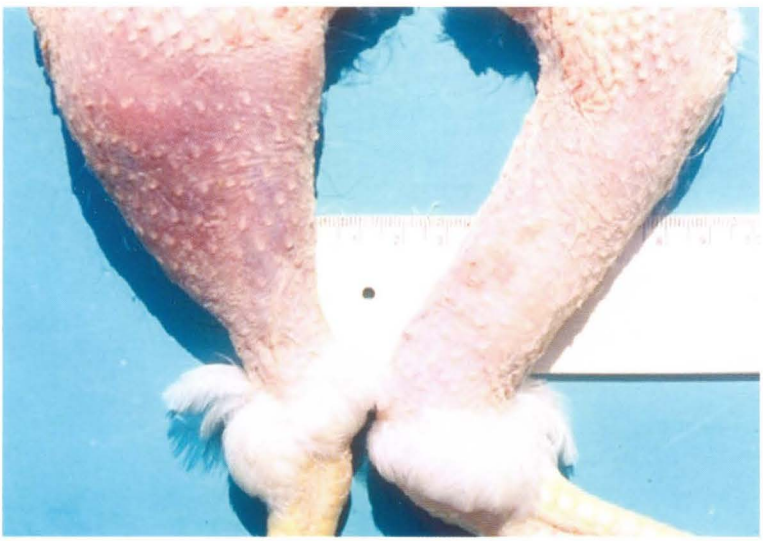
RUPTURE OF THE GASTROCNEMIUS TENDON IN BROILER BREEDERS



490. This state could cause significant economical losses in broiler breeder flocks. It is usually seen in birds older than 12 weeks but is also observed in breeders at the age of 7 weeks or older than 24 weeks. The rupture could be uni- or bilateral. Clinically, lameness is observed. In affected birds, swelling of the posterior surface of the leg, just above the tibiotarsal joint could be palpated.

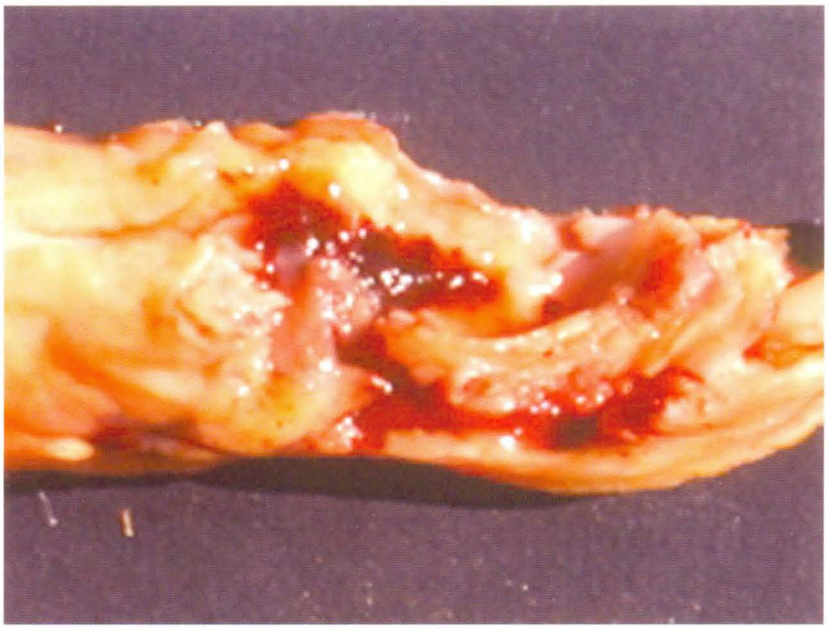


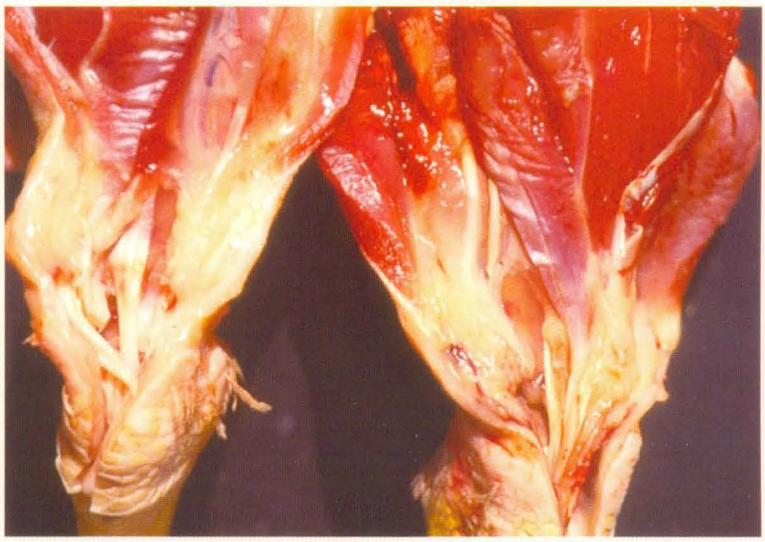
491. In acute lesions, haemorrhages are visible through the skin. In older lesions, a blue-greenish discoloration is present whereas in chronic lesions, very hard masses as abnormal subcutaneous tissue are palpated.

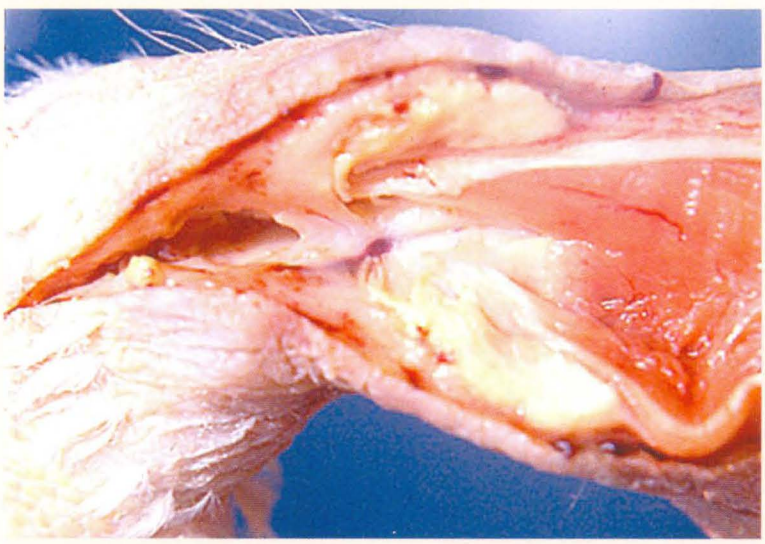


492. In part of birds, the muscles of affected legs undergoes a various degree of atrophy. In acute lesions, swellings and haematomas under the skin of the posterior surface of the leg are detected.

493. The rupture is usually seen as irregular, transverse interruption just above the tarsal joint. Among the haematoma, the free end of the tendon could be seen.

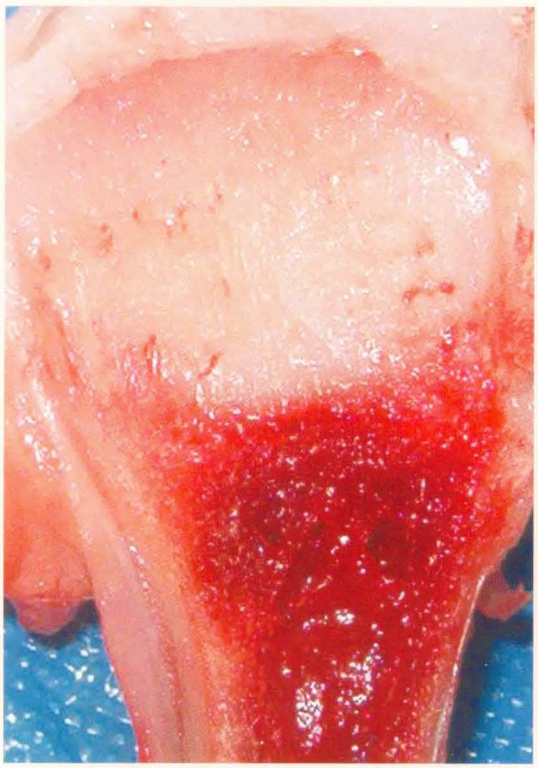




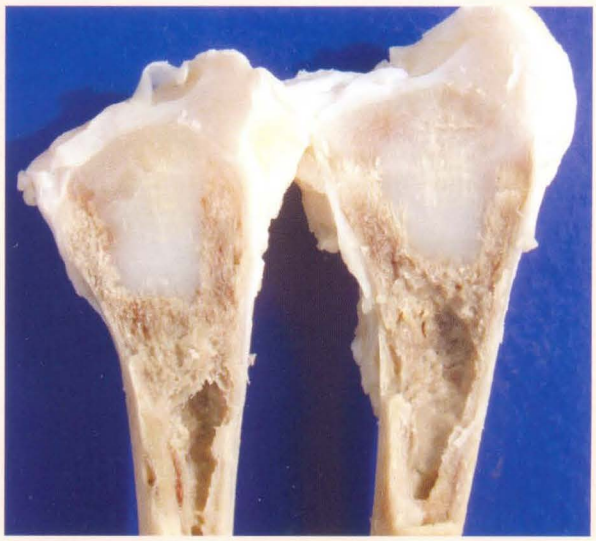


494, 495. In older or chronic lesions, the blood is partially or completely reabsorbed. The end of ruptured tendon and the adjacent tissue are involved at a various extent by a newly grown fibrous tissue. RGT should be differentiated from reovirus- and MS infections, where the gross and histological lesions are with a marked inflammatory character

DYSCHONDROPLASIA



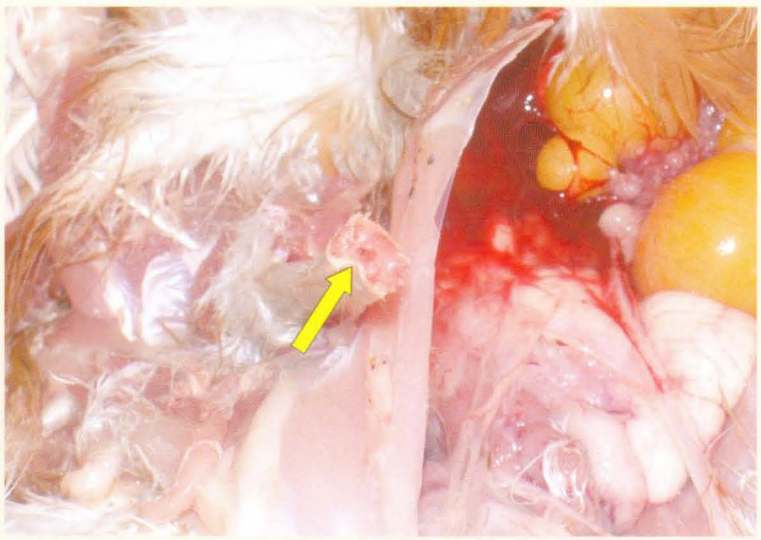
496, 497. Dyschondroplasia is a defect of the growth plates in meat type poultry. It is characterized by abnormal cartilage masses under the growth plates of long bones. Most commonly, it is established in the proximal tibiotarsus and that is why it is called tibial dyschondroplasia. Dyschondroplasia could be also seen in the proximal and distal femur, the distal tibia and the proximal humerus. Clinically, affected chickens exhibit reluctance to move and lameness. Fractures could also be seen. The aetiology of dyschondroplasia is related to genetic factors, rapid growth in broilers, vitamin D metabolism, mycotoxins etc.

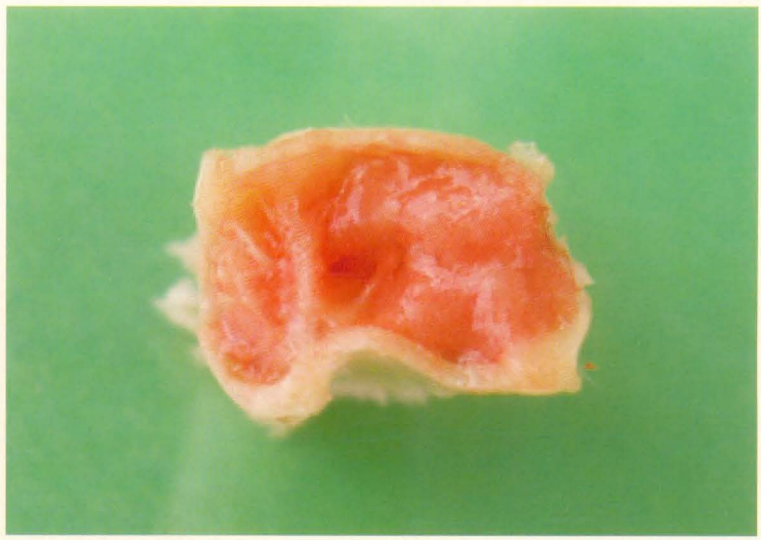


CAGE LAYER FATIGUE

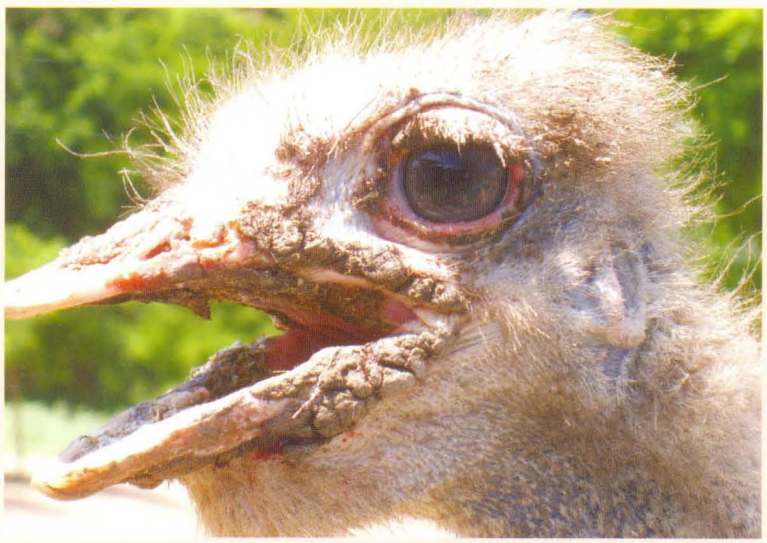
498. The cage layer fatigue syndrome in birds is characterized by an inability to stand on their feet and fragile bones. It is mainly observed in young layer hens reared in batteries in the period of maximum egg-laying. Affected birds lie down and stopped eating. Egg shells become thin.

499, 500. The calcium deficiency is layer hens results in initial removal of calcium from bones, to complete depletion of the medullary bone and thereafter, of the bone wall. The bones are strongly thinned and spontaneous fractures, especially of the tibia and the femur could occur. Although the severe calcium deficiency is often a triggering factor, the aetiology of the syndrome seems to involve other, yet unknown factors. The supplementation of calcium, phosphate and multivitamin preparations in the diet and rinking water, the regulation of avian population density into cages and ensuring adequate nutritional and drinking fronts are also contributing for the favourable outcome of the condition.

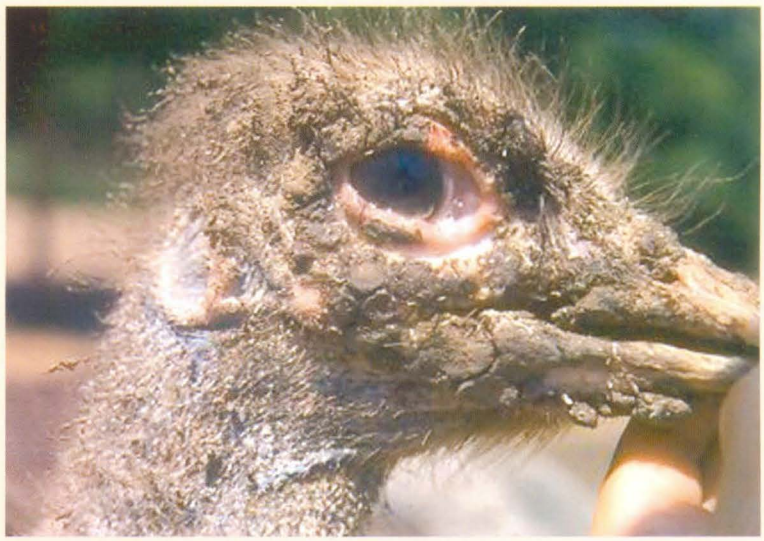




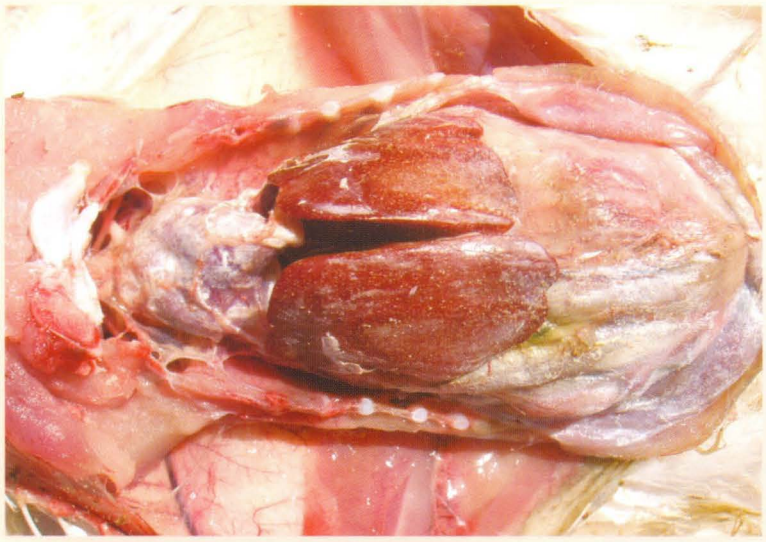
HYPERKERATOSIS



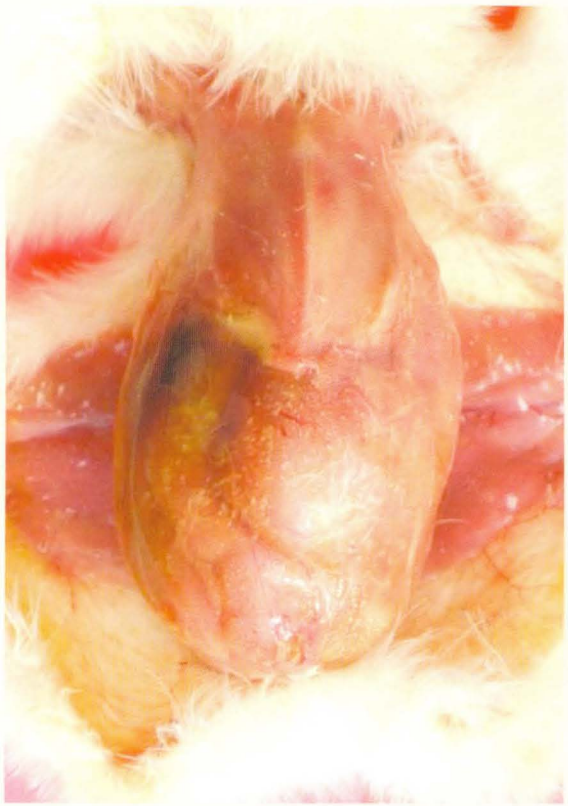
501, 502. A local form of hyperkeratosis in an ostrich. Increased amount of keratin in the horny tissues - down, feathers and beak - is present, resulting in their coarse appearance. The state is linked to impaired metabolism of sulfur-containing amino acids, vitamin A deficiency etc.



GOUT

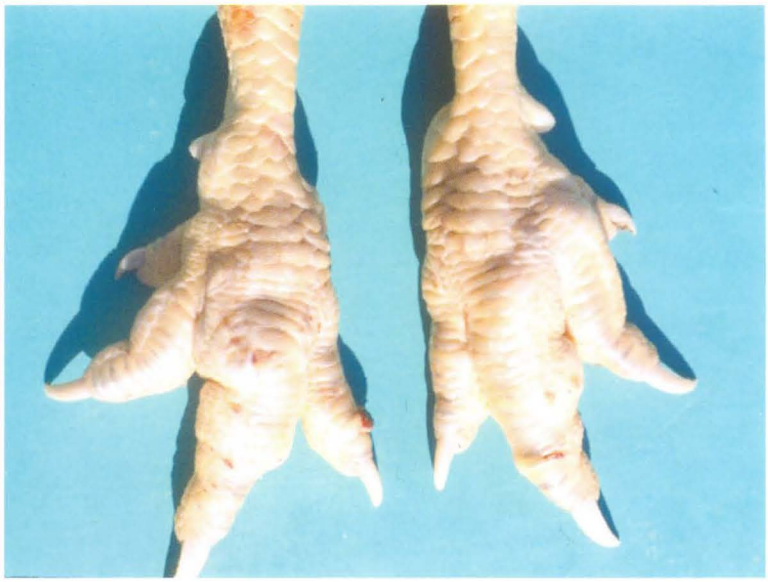


503, 504, 505. The gout is characterized by retention and buildup of urates in tissues. It is seen as two separate syndromes: visceral gout and articular gout. The **visceral gout** is manifested with deposition of urates in renal tubules and the serous coats of the heart, the liver, the mesentery, the air sacs or the peritoneum. The urate deposits on serous coats resemble a chalky white dust. Visceral urate deposits are generally due to renal failure. Possible causes for this could be obstruction of ureters, renal damage or dehydration.

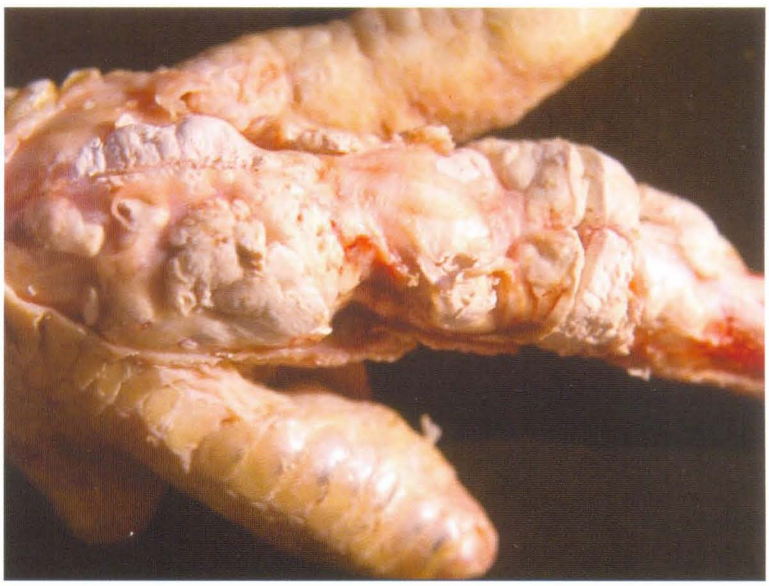


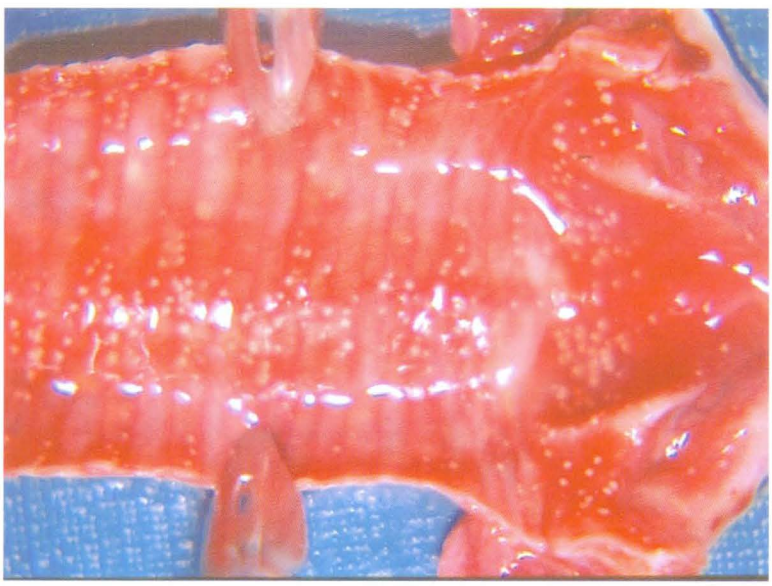
506, 507. Most commonly, visceral gout following dehydration is observed in newly hatched chickens after overheating or a more prolonged stay in the hatchery. Visceral gout outbreaks are related to vitamin A deficiency, treatment with sodium bicarbonate, mycotoxicoses etc.

508. The articular gout is characterized with periarticular urate deposits (tophi), especially around the joints of toes and the foot. The joints are enlarged and toes - malformed.



509. After opening of affected joints, the periarticular tissue is white. A white semi-liquid matter, due to urate deposits, could also be seen.

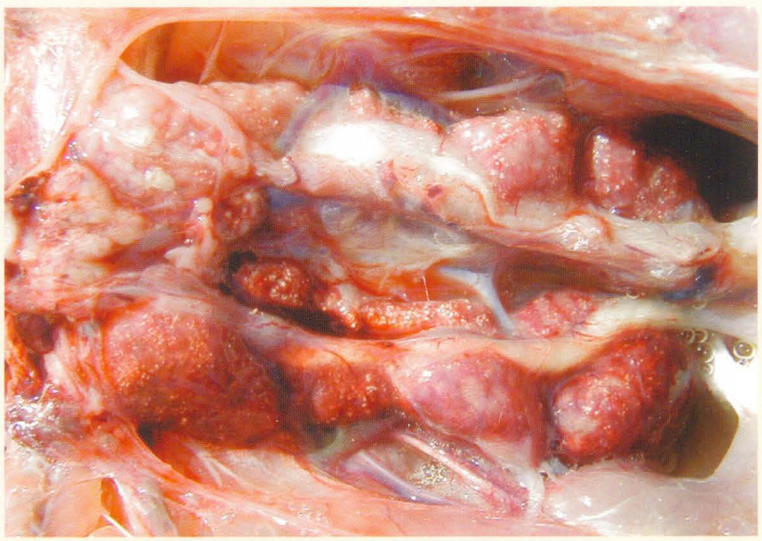


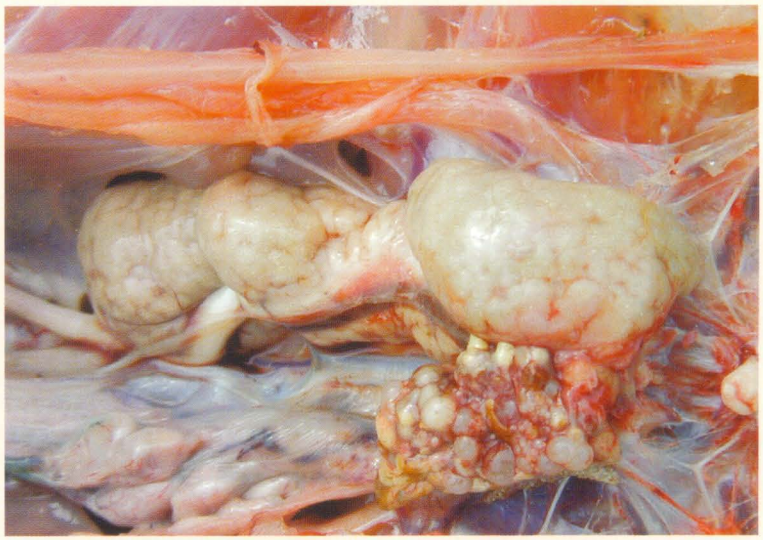


510. In chronic cases, urate precipitations could be observed in the trachea, the comb, wattles etc.

511, 512.

Urolithiasis is an aetiologically unknown state, occurring primarily in cage layer hens, characterized by obstruction of one or both ureters with urates, atrophy of one or more renal lobes and a various degree of renal and visceral gout. A number of aetiological factors are related to this condition: protein excess, calcium excess (3% or more), sodium bicarbonate toxicity, mycotoxins (ochratoxin etc.), vitamin A deficiency and nephrotropic strains of the infectious bronchitis virus. The lower phosphorus levels (under 0.6%) are probably helping the manifestation of the disease. There are no specific clinical signs except for the depression and the weight loss. The death rate could increase and persist around 2-4% monthly during the productive period. The total mortality is heavily affected flocks could reach 50%.





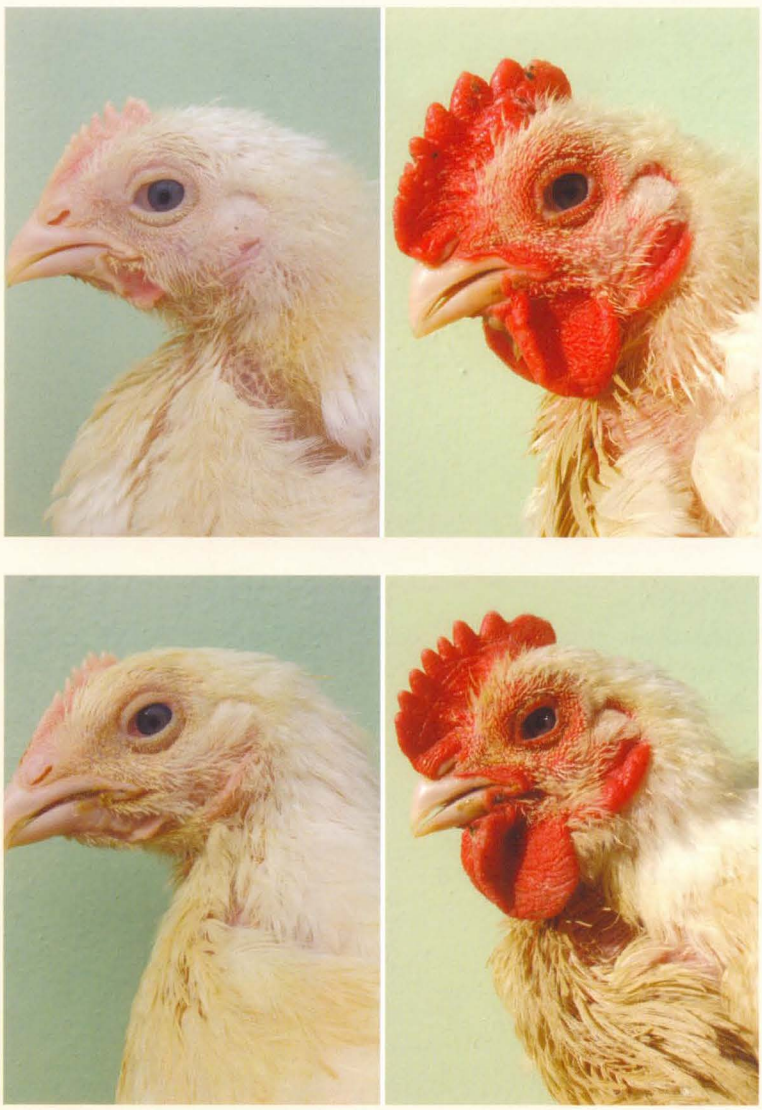
HYPERANDROGENISM IN BROILER CHICKENS

513*, 514*.

Hyperandrogenism in broilers is a condition, manifested with marked signs of masculinization in chickens from both genders. The first signs could start very early after the age of 10-12 days. A very noticeable reddening of the comb and wattles, coarse feathering of the face, strong growth of nails and a highly aggressive behaviour in about 100% of birds are observed.

515. Facial wounds following manifestations of aggressive behaviour.

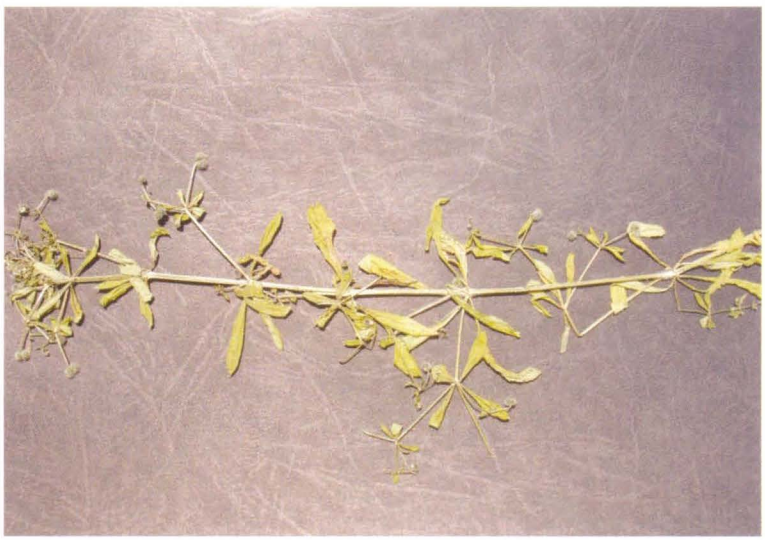
516*, 517*. The peak of clinically manifested masculinization traits is between the age of 20 and 30 weeks in all chickens. The serum testosterone is manifold higher. In 23-day-old broilers, average testosterone concentrations were 709 ng/dl vs 36 ng/dl in intact chickens at the same age. The mycotoxins following contamination of some of forage components with moulds are assumed to be a possible cause for this state, but yet, it is not clear if this or another factor is causing hyperandrogenism.



* Comparison of exterior signs between intact (left) and affected (right) chickens at the same age.

518. General view of 35-day-old broiler chickens with signs of hyperandrogenism. All chickens in the premise are with strongly developed secondary sexual characteristics.

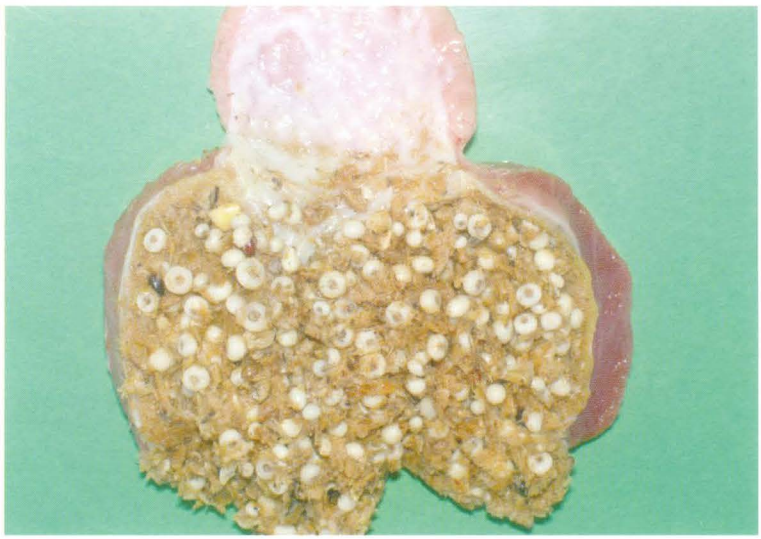
EFFECT OF CLEAVERS (GALIUM APARINE) SEEDS ON HEALTHY STATUS AND PRODUCTION TRAITS IN BROILER CHICKENS



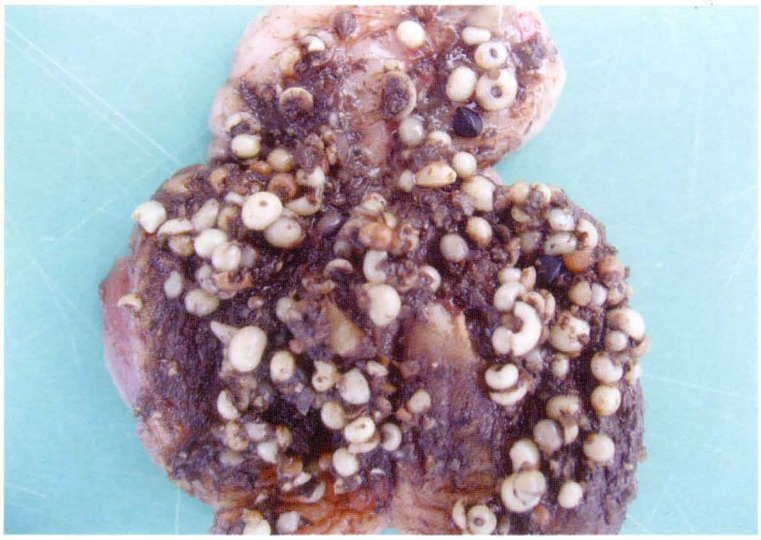
519. Galium aparine, cleavers or stickywilly, is an annual weed in cereals and earthed-up crops, is an herbal plant belonging to the *Galium* genus (herbarium).

520. The seeds (fruit) are dry, round or kidney-shaped, with smooth, corrugated or granular surface, sometimes covered with hooked prickles, brown or grey colour and are notched on one side („nuts"). On the left - seeds with coat, on the right without coat.

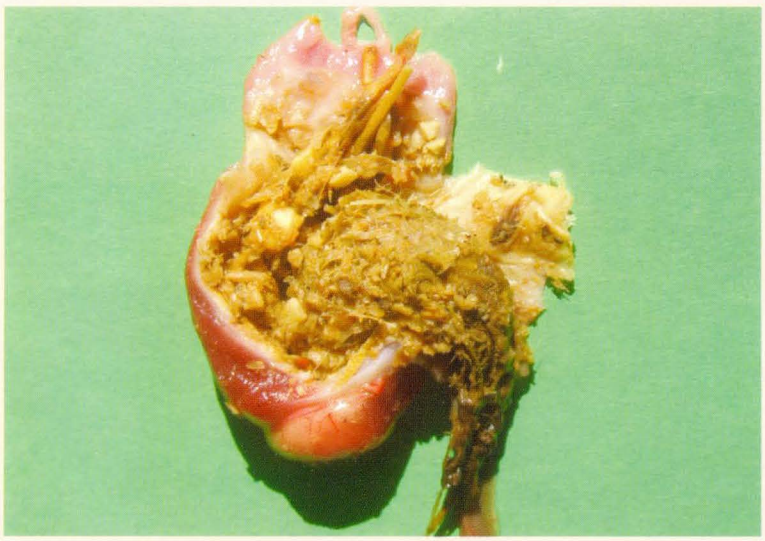
521. In the gizzard of chickens, the seeds become white with a contrasting black-coloured unipolar groove, similar to an eye. Most probably, that is why they are called **„snake eye".**



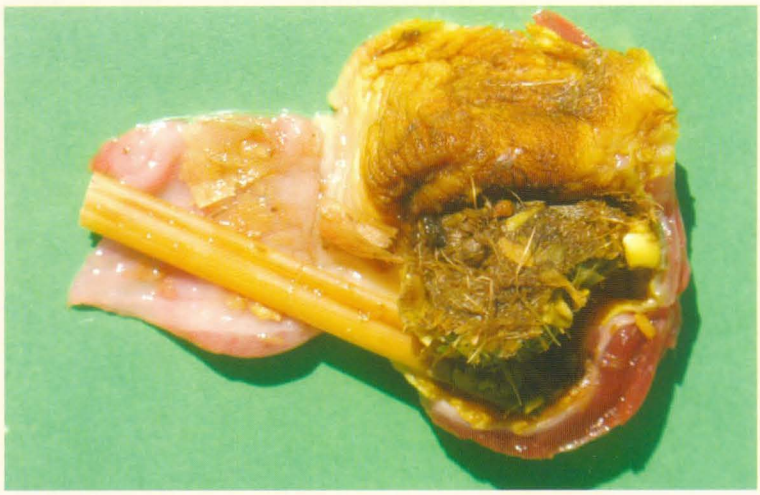
522. Purulent necrotic processes in the gizzard of a chicken. The health is influenced by consequences of mechanical obstruction because of seeds' accumulation in the gizzard. A direct toxic effect of Galium aparine is also supposed (primarily hepato- and nephrotoxic), due to release of toxic glycosides and other toxic substances from the seeds.

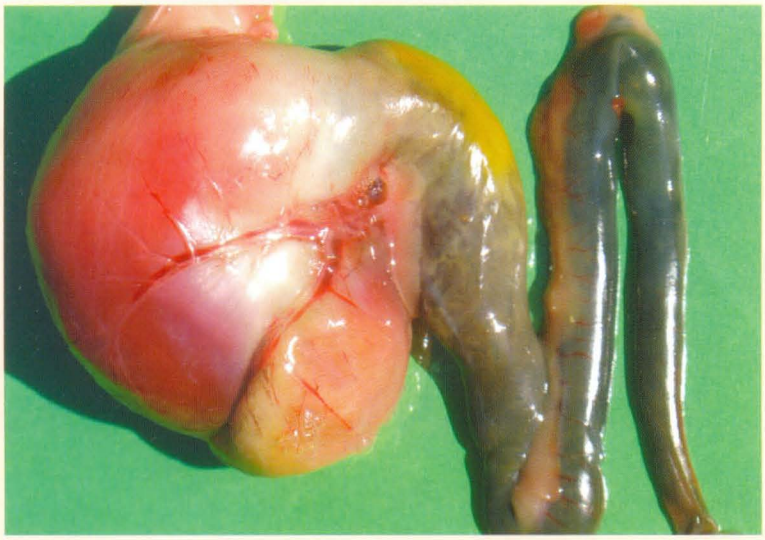


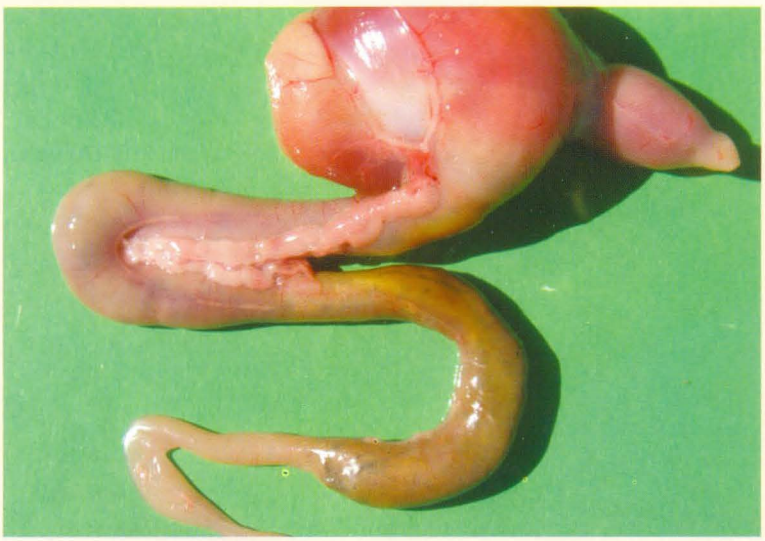
GIZZARD IMPACTION IN TURKEY POULTS



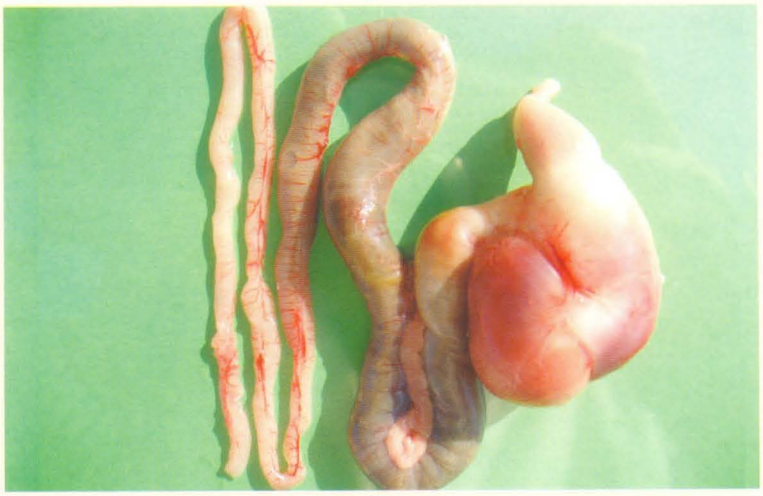
523, 524. The gizzard impaction could results in high mortality in turkey poults during the first three weeks of life. Affected turkey poults are dehydrated, with empty gut, but the gizzard is filled with hard fibrous masses.



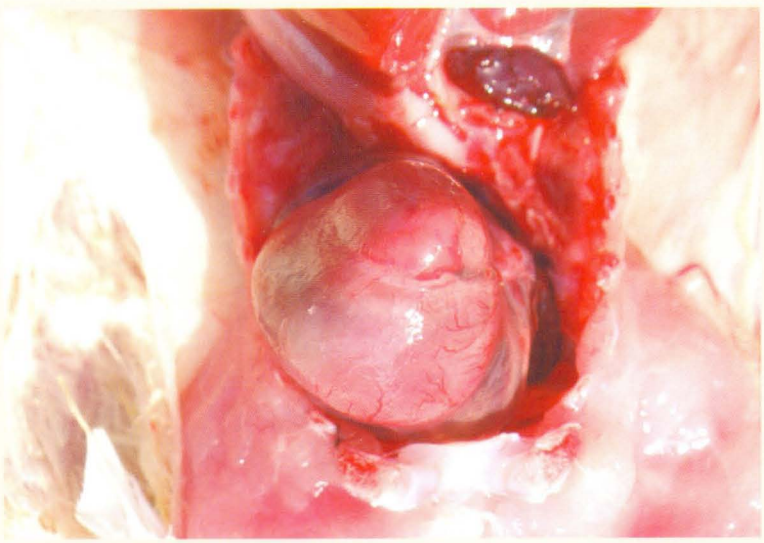


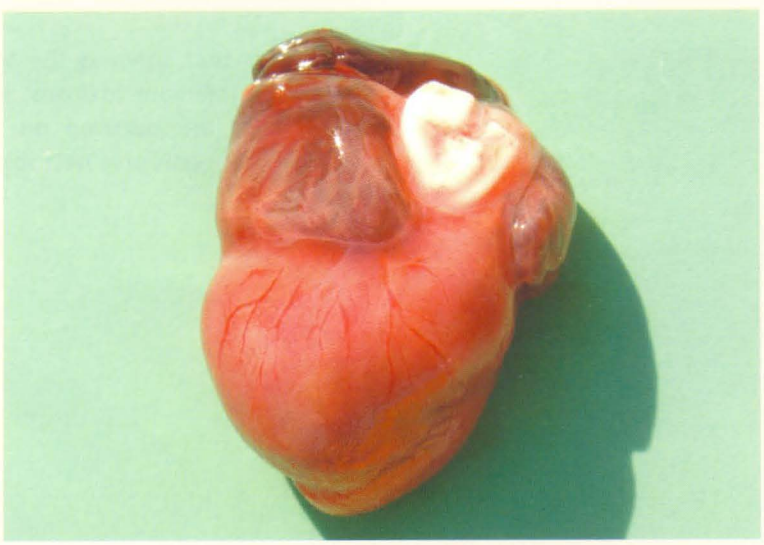


525, 526, 527. In some cases, the fibrous masses enter the first part of the duodenum or the lower parts of the small intestine. The impaction is resulting from eating litter that the gizzard cannot ingest. The prevention is aimed at control on litter eating.

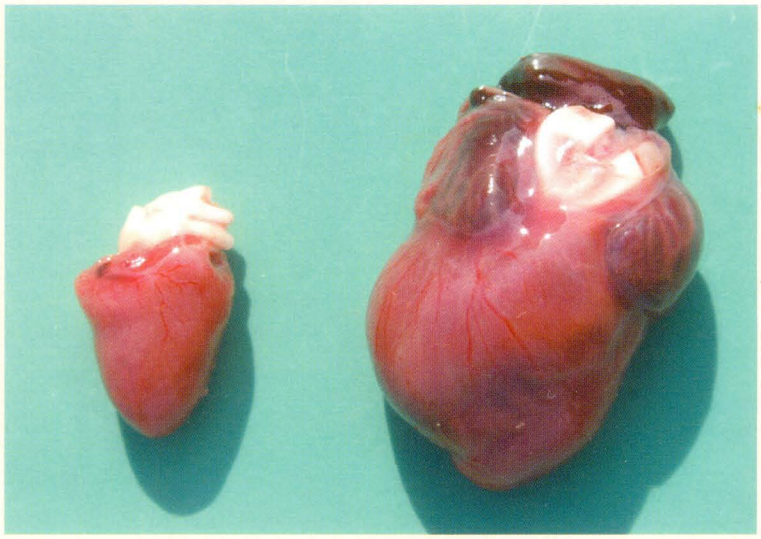


ROUND HEART IN TURKEYS (DILATED CARDIOMYOPATHY)

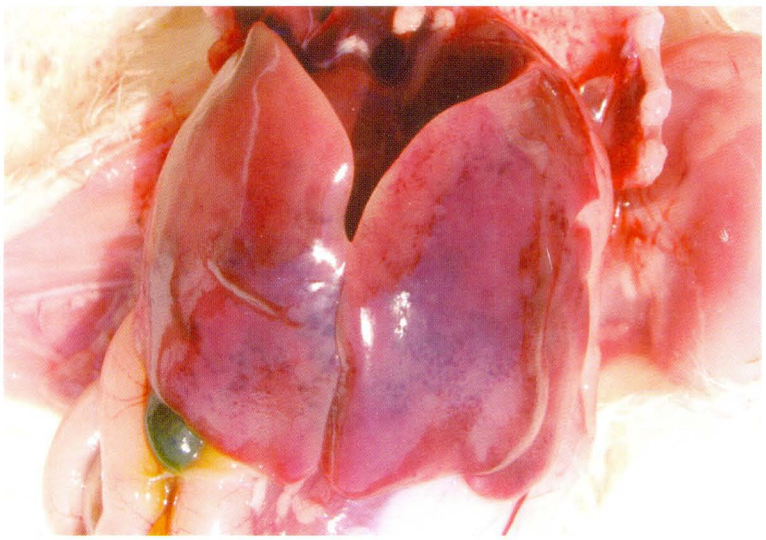




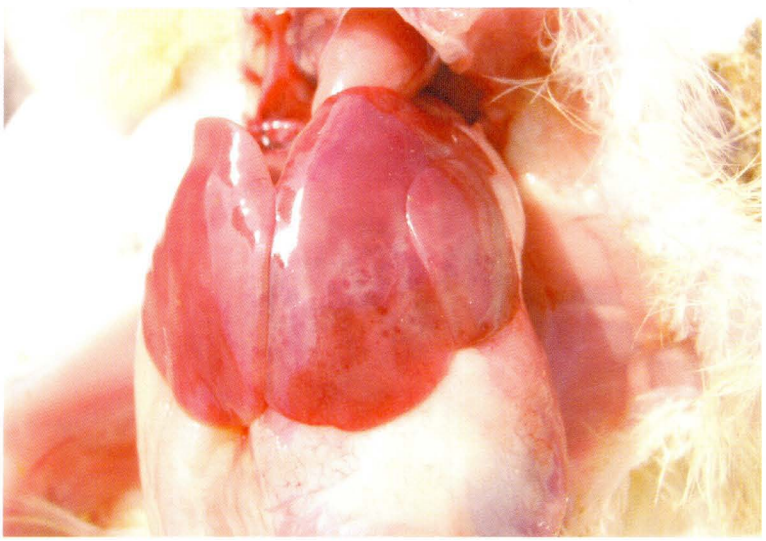
528, 529, 530. The aetiology is not known, but genetic factors or early viral myocarditis is supposed. The state is lethal in turkeys at the age of 1 - 4 weeks. Pathoanatomically, a severe dilated cardiomyopathy, often accompanied with ascitis, hydropericardium and congestion of other organs is detected.



ACUTE SELENIUM INTOXICATION

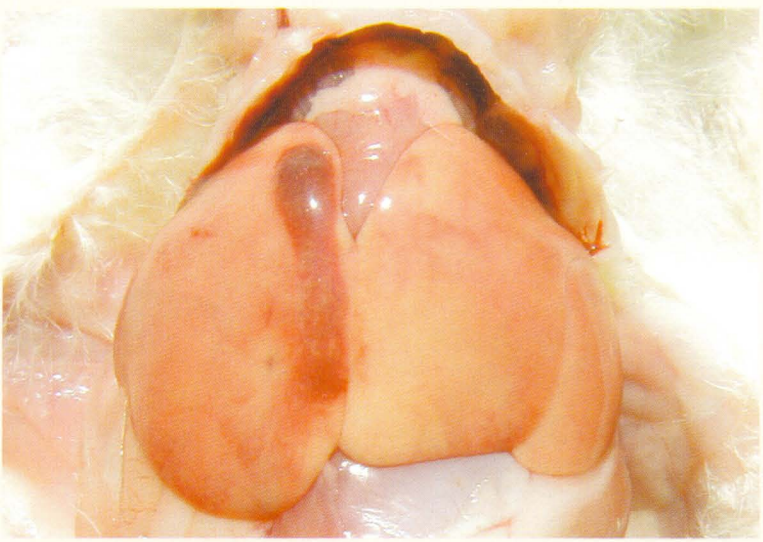


531. Acute selenium intoxication in birds is usually seen after multifold overdosing of preparations containing inorganic selenium with the aim of prevention or therapy. In the early hours after intake of toxic doses of selenium, a high death rate (up to 100%) and massive haemorrhages in the liver are seen.



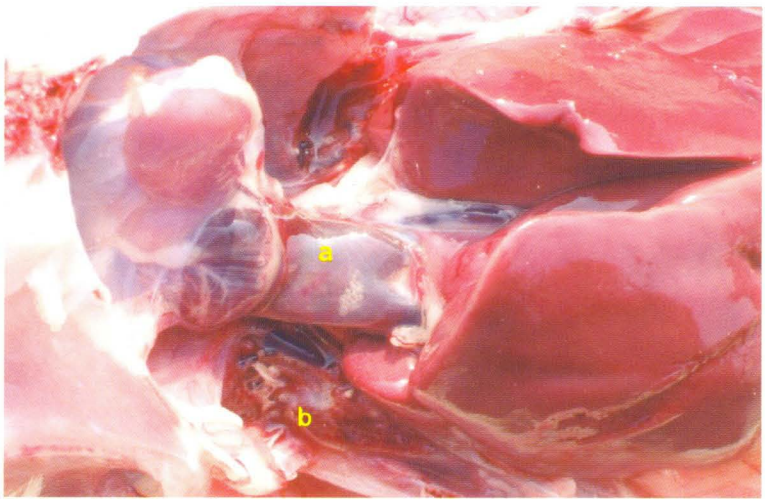
532. In birds that survived the later stage of the selenium toxicosis, liver haemorrhages are outlined on the background of dystrophic necrobiotic changes.

ACUTE PROPANE-BUTANE INTOXICATION



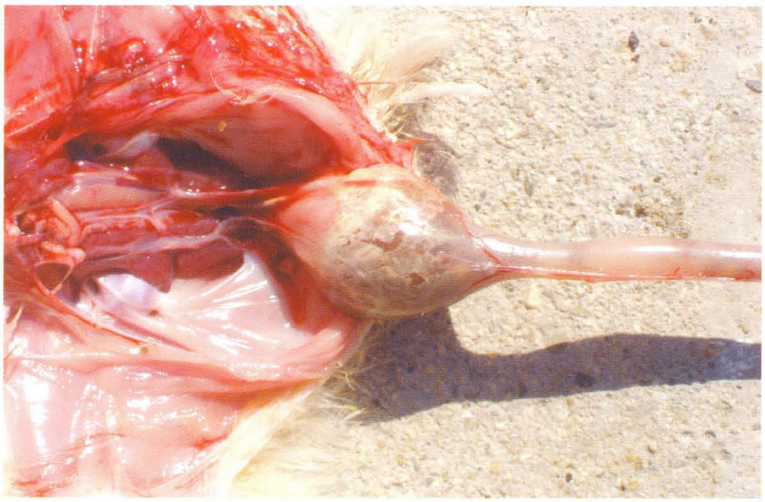
533, 534, 535. In cases of acute intoxication with propane butane (due to damage in heating appliances where propane-butane is used as energy source for heating), asphyxia, cyanosis of the featherless skin, pulmonary oedema and subcapsular haemorrhages in the liver are observed.

OVERHEATING AND ASPHYXIA



536. Stasis in the right half of the heart, overfilling of the posterior vena cava (a) and oedema of lungs (b) following overheating. When the temperature in the premise reaches 35°C, the cooling in birds begins by convection, conduction and radiation as they have no sweat glands. The excessive respiration results in oversaturation with CO_2 and then, in asphyxia.

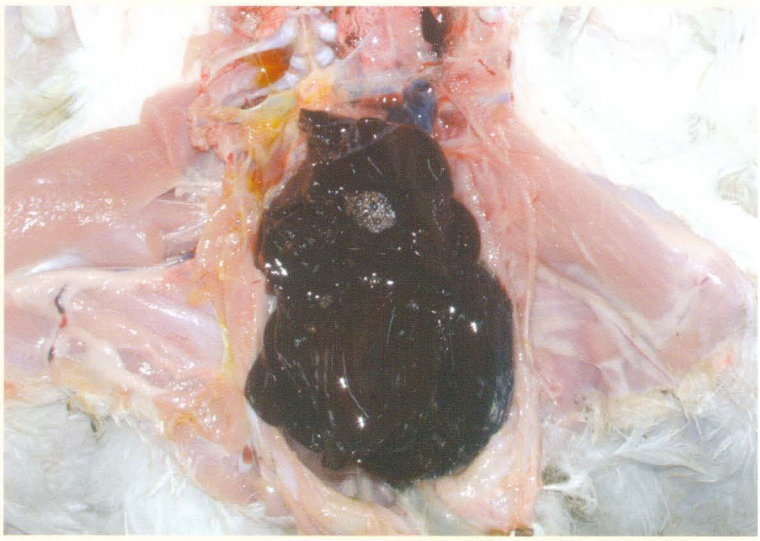
PASTED VENT

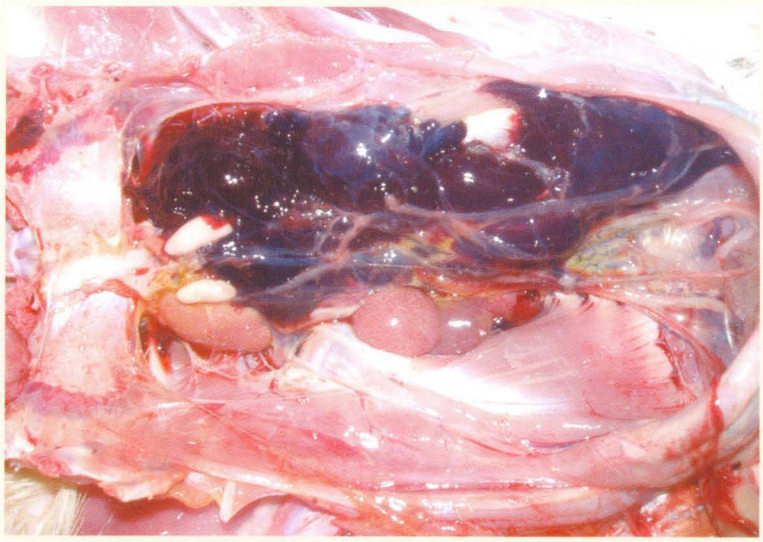


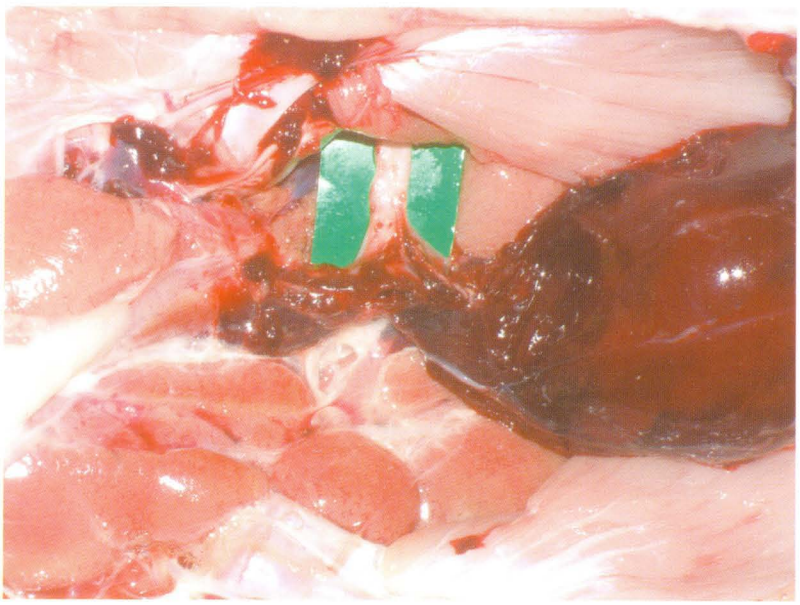
537. Pasted vent is usually observed in diarrhoea, when the rectal masses are sticked to adjacent feathers. The observed plugs prevent the evacuation of the next faecal masses. This results in dilatation and obstipation of the rectum.

SPONTANEOUS RUPTURE OF THE CAUDAL RENAL ARTERY IN TURKEYS

538, 539, 540.
A case of spontaneous rupture of the caudal renal artery in broiler turkeys is observed at the age of 8 - 12 weeks. Some birds in an excellent body condition die. The carcasses are anaemic. The necropsy shows that in some birds, the entire body cavity is filled with clotted blood (538) and in others - massive subcapsular coagula from the side of the affected kidney are detected (539 and 540).

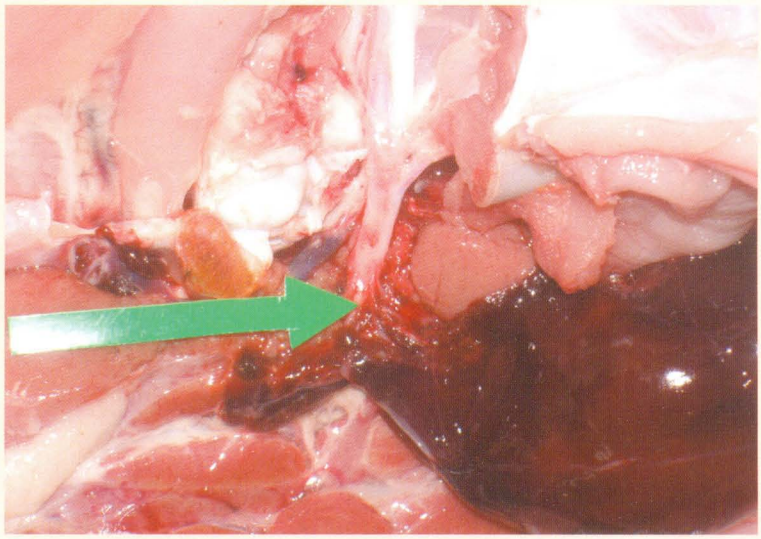


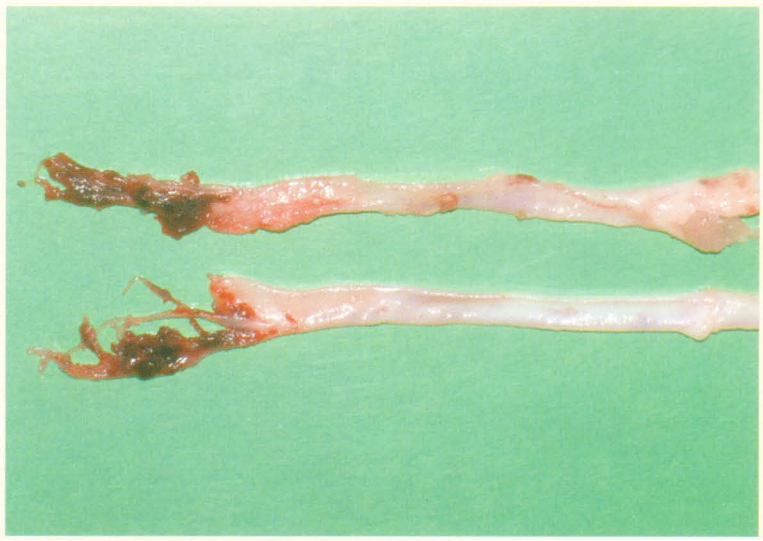




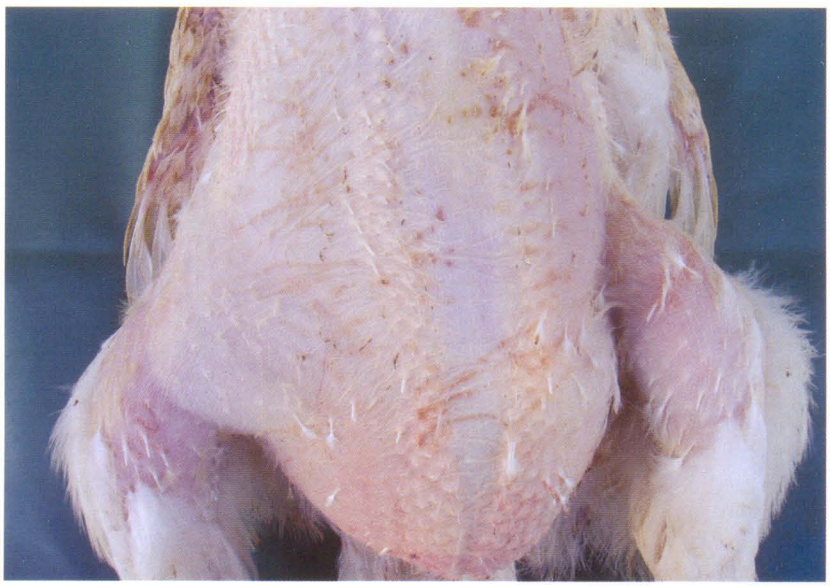
541. In all cases, unilateral rupture was observed, primarily of the left renal artery.

542, 543. After careful separation of the renal parenchyma, the torn ends of the artery are revealed. The cause is unknown but some accompanying pathogenetic factors could be the high blood pressure in turkeys, their natural predisposition to atherosclerosis and the lack of intramural vasa vasorum of the descending aorta.





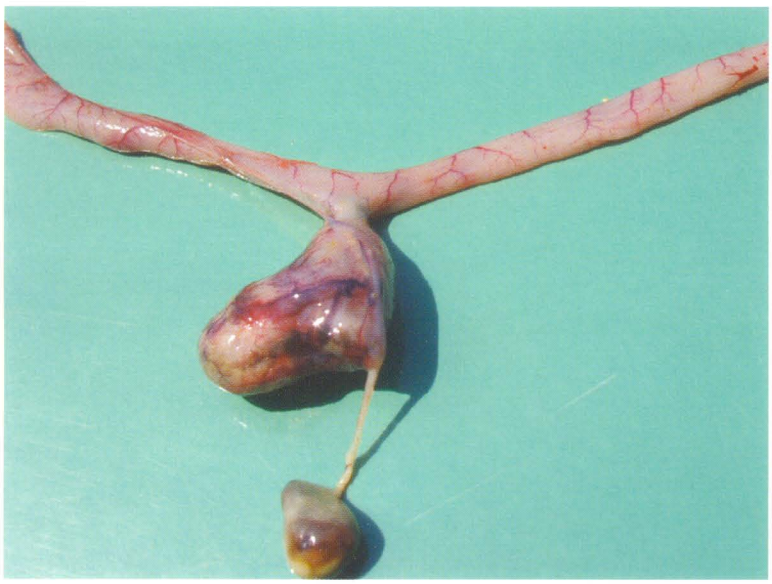
SUBCUTANEOUS EMPHYSEMA



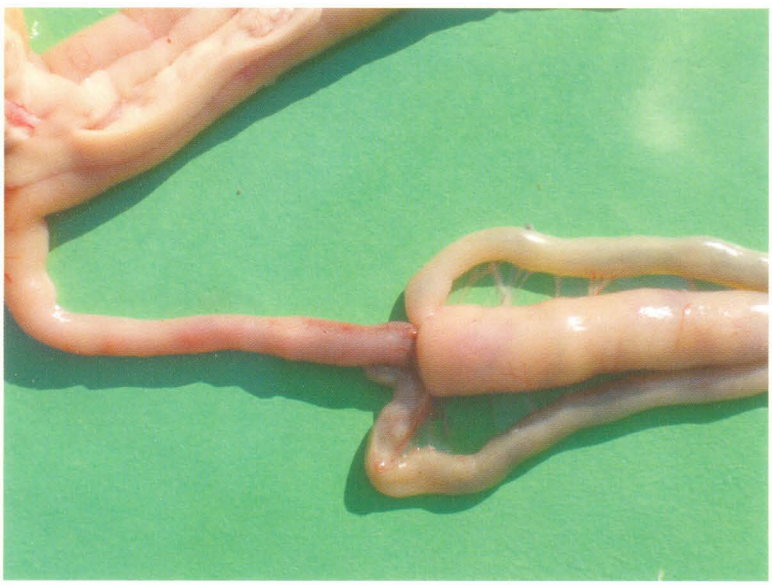
544. Subcutaneous emphysema in the right inguinal region. It occurs after penetration of air or gases in the subcutaneous connective tissue. Usually, it results after air-sac tearing or skin injury.

545. Subcutaneous emphysema in the region of the head.

GASTROINTESTINAL IMPACTION



546. DIVERTICULUM.
A sacciform blind pouch occurring after attachment of the intestinal wall to an unabsorbed yolk sac, causing impaction.

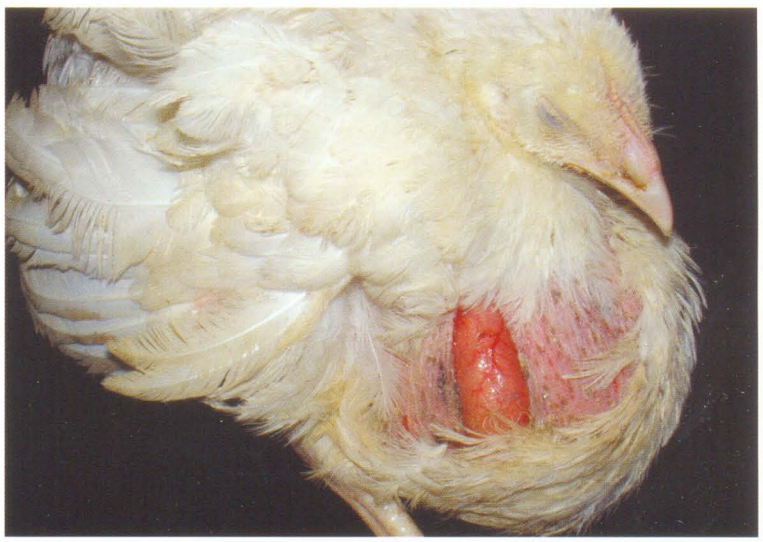


547. INVAGINATION.
Involvement of anterior parts of the small intestine, the mesentery and the tops of the caecum into the ileum. Usually, it occurs with a strong intestinal peristalsis, often following ingestion of feed after a restrictive feeding regimen.

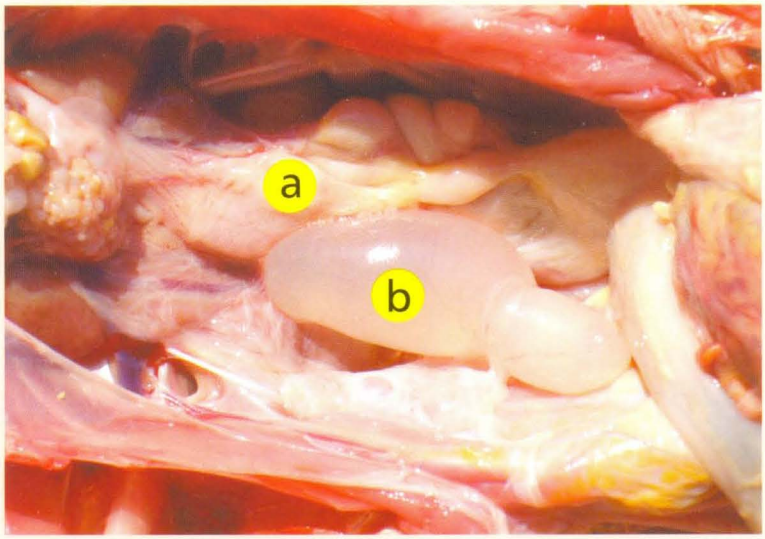
548. VOLVULUS.
Rotation of the small intestine on the longitudinal axis of the mesentery, resulting in venous stasis and necrosis of the intestinal wall.

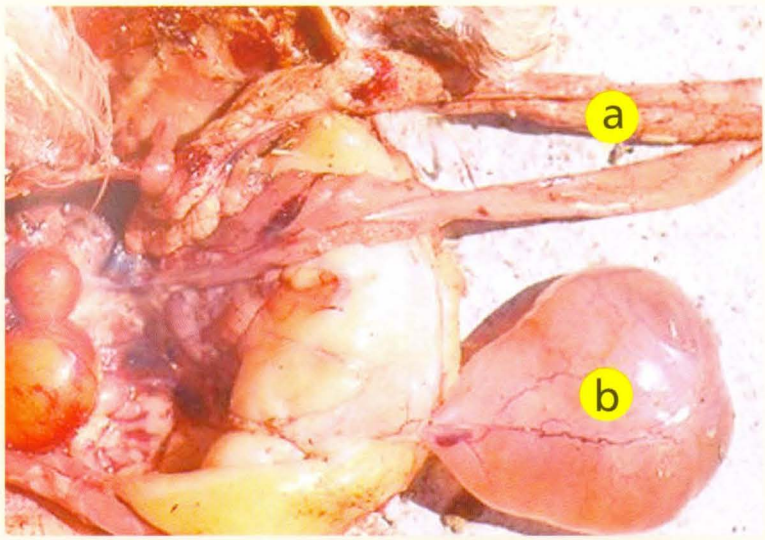
549. CROP IMPACTION.
It is caused by hard, fibrous feed or litter, whose accumulation results in impaction.

550. CROP IMPACTION.
The retained content into the crop sometimes causes putrefactive necrotic processes, affecting the crop wall and the covering skin.



MALFORMATIONS





551, 552. PERSISTENT RIGHT OVIDUCT. If the regression of the right Mullerian duct, that is embryonally present in female chickens as oviduct, does not occur, a cystic dilatation of this structure is seen, whose size varies from a long cystic formation to a big sac, filled with fluid:
a/ functional left oviduct
b/ persistent right oviduct

553. ABSENCE OF EYES (ANOPHTHALMIA) AND CRANIAL MALFORMATIONS are frequent malformations in newly hatched chickens.

554. OPEN BODY CAVITIES. An additional skin formation (pouch) to the left laterocaudal abdominal wall, with part of the abdominal organs found within.

555, 556. POLYPODIA. The occurrence of more legs than normal is often seen in broilers. Such birds frequently survive up to the end of the fattening period.

Abbreviations

AAS - avian adenovirus splenomegaly

AAVs - avian adenoviruses

AEV - avian erythroblastosis virus

APV - avian pneumovirus

CAH - cholangiohepatitis

CIA - chicken infectious anemia

DPM - deep pectoral myopathy

EDS 1976 - egg drop syndrome 1976

ER - erythroblastosis

FP - fowl pox

GD - gangrenous dermatitis

HE - hemorrhagic enteritis

HEV - HE virus

HVT - turkey herpesvirus

IB - infectious bronchitis

IBH - inclusion body hepatitis

IBD - infectious bursal disease

IBDV - IBD virus

IEM - infectious encephalomyelitis

LL - lymphoid leucosis

LT - laryngotracheitis

MC - myelocytomatosis

MD - Marek's disease

MDV - MD virus

MG - mycoplasma gallisepticum

MS - mycoplasmasynoviae

MSDV - marble spleen disease

ND - Newcastle disease

NE - necrotic enteritis

PHS - pulmonary hypertension syndrome

RGT - rupture of the gastrocnemius tendon

SHS - swollen head syndrome

TRT - turkey rhinotracheitis

UE - ulcerative enteritis

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CEVA SANTE ANIMAL HEALTH

INJECTABLE ANTIBIOTICS

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
INTRAMICINE 100 mL, 250 mL QD for 3-5 days	Benzylpenicillin procaine 20 millions ME/ Dehydrostreptomycin - 20 g/ 100 mL	CTL, HRS: 5 mL/100 kg; young: 8 mL/100 kg.; SHE, GOA: 3-5 mL/per animal; PIG: 1 mL/10 kg, over 130-150 kg - 5 mL/100 kg; DOG, CAT: 0.5 mL/5 kg	CTL, HRS, SHE, GOA, SWI, PIG, DOG, CAT
GALLIMYCIN 200 50 mL QD for 3-days	Erythromycin base - 200 mg/mL	CTL,CLF: 0.5-1 mL/ 50 kg; SWI: 0.5-1.5 mL/ 50 kg; PIG: 0.5 mL/ 5 kg; SHE: 0.5-1 mL/50 kg; LMB: 0.25 mL/5 kg	CTL, SWI, SHE
SPECTAM 100 mL QD for 3-5 days	Spectinomycin – 10 g / 100 mL	CTL, HRS, SWI: 10-20 mL/100 kg; CLF, SHE: 10-15 mL/50 kg; suckling PIG: 0.5 mL/animal; POU: 0.1-0.2 mL/kg; DOG and CAT: 1 mL/5 kg.	CTL, HRS, SWI, SHE, GOA, POU, DOG, CAT
TETRAVET LA 100 mL, 250 mL depot 72 h every 72 h	Oxytetracycline - 20 g/100 mL	CTL, SWI, SHE and GOA - 1 mL/10 kg.	SWI, SHE, GOA, CTL
VETRIMOXIN LA 100 mL, 250 mL depot 48 h every 48 h	Amoxycillin – 15 g / 100 mL	COW, SHE, PIG, SWI: 1 mL/10 kg; suckling PIG: 3rd-5th day - 1 mL/pig i.m. for prevention of diarrhoea	SWI, SHE, GOA, CTL

ORAL ANTIBIOTICS

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
COLIVET 20 kg, 1 kg, 100 g	Colistin - 120 millions ME/100 g (6 %)	1 g/1 L water therapeutically from 5 to 7 days; 1-2 kg/1 tonne forage 5 - 7 days	CLF, PIG, POU, RBT
COLIVET SOLUTION 1 L, 5 L	Colistin - 200 millions ME/100 mL	5-6 mL/10 L water for 5 days - POU, RBT; 6-7 mL/10 L water for 5 days - PIG, CLF	POU, RBT, PIG, CLF
DOXYVIT PREMIX 10 kg, 1 kg	Doxycycline - 100 g/1 kg Vit. C - 20 g/1 kg	1 - 2 kg /1 tonne forage - 5-10 days	SWI, CTL, POU
DOXYVIT POWDER 10 kg, 1 kg, 100 g	Doxycycline - 100 g/1 kg Vit. C - 80 g/1 kg	1 g/1 L water - 3 to 5 days	POU, CLF, SWI
DOXYVIT 50% WSP 10 x10 g, 100 g,1 kg,10 kg	Doxycycline - 50 g/100g, Citric acid - 5 g/100 g	POU: 2-3 g/10 L water for 5 days; SWI: 2 g/10 L water for 8 days	SWI, POU
FLUMISOL 10% 1 L	Flumequine - 10 g/100 mL	PIG: 1.5 mL/10 kg; CLF, SHE, GOA, POU: 1.2 mL/ 10 kg – 3 to 5 days	POU, PIG, CLF, SHE, GOA, RBT
FLUMIQUIL 50% 25 kg, 1 kg, 100 g	Flumequine - 50 g/100 g	POU: 1 g/5 L water; SWI: 3 g/100 kg ; CTL, SHE, GOA: 2.4 g/ 100 kg ; FIS: 2.4 g/100 kg 3-5 days	POU, PIG, CLF, SHE, GOA, FIS
GALLIMYCIN PULVIS 25 kg, 2 kg, 200 g	Erythromycin thiocyanate - 5.51g/100g	POU, RBT: therapeutically: 2-4 g/1 L water; CLF: 5-10 g/50 kg BID therapeutically 3 to 5 days.	POU, CLF, RBT
OTC 50% premix 30 kg	Oxytetracycline - 500 g/1 kg	0.51 kg/1 tonne forage from 7 to 10 days.	SWI, PIG, CLF, POU
TIACLOR PREMIX 25 kg, 5 kg, 1 kg	Tiamutin 50 g + Chlortetracycline 200 g/1 kg granulate	2 kg/1 tonne forage - 7 days for prevention; 4 kg/1 tonne forage - 7 days therapeutically	SWI, PIG
CEVALIN PREMIX 25 kg, 10 kg, 5 kg, 1 kg	Lincomycin HCl 110 g/1 kg	SWI: dysentery: prevention 1 kg/2.5 tonnes forage for the entire risk period; treatment: 1 kg/1 tonne forage as basic diet for 3 weeks; Mycoplasma pneumonia: 2 kg/1 tonne forage for 3 weeks; ileitis 400 g to 1 kg/1 tonne forage or 0.3-0.4 kg premix/tonne forage for 5-7 consecutive days	SWI
CEFLOR 10% LIQUID 5 L, 1 L, 100 mL	Florfenicol 100g/1 kg	SWI: 1.5-2 mg/100 kg daily for 5-7 consecutive days or 0.3-0.4 kg premix/ tonne forage for 5-7 consecutive days	SWI
CEFLOR 10% PREMIX 20 kg, 10 kg, 5 kg, 1 kg	Florfenicol 100 g/ 1 L	SWI: 1.5-2 mL/100 kg for 7 days; POU: with drinking water, < age of 4 weeks 100 mL/100 L water for 5 days; > age of 4 weeks - 200 mL/100 L water for 5 days	
SPECTAM SH 100 mL, 1000 mL	Spectinomycin - 5 g/100 mL	1 dose = 1 mL/1 animal BID, 3-5 days	PIG, LMB, GOA
VETRIFLOX 200 1 L	Norfloxacin - 20 g / 100 mL	POU: 1 mL/2 L water for treatment. PIG: 0.25 mL/1 pig up to 5 kg	POU, PIG
QUINOEX 10 % 1 L	Enrofloxacin - 100 g/1L	POU: 1 mL/2 L water daily for 3-5 days	POU
VETRAMOX 80% pow 1 kg, 200 g	Amoxycilin -80 g / 100 g	POU: 10-20 g/100 L water. PIG: 10-20 g/50 kg forage, 3-5 days	POU, SWI
VETRIMOXIN 50 x 1kg, 100 g (10x10 g)	Amoxycilin - 50g / 100 mL	POU: 10-20 g/100 L water PIG: 15-30 g/100 L or 50 kg forage	POU, CLF, PIG
QUINOCOL sol. 1 L	Enrofloxacin + Colistin	1mL/2 L water daily for 3-5 days	POU
TIAMVET 800 x 25 kg, 1 kg	Tiamulin HF - 800 g/1 kg	80 - 200 ppm/1 tonne forage	SWI

TIAMVET 45 x 1 kg water-soluble	Tiamulin HF - 450 g/1 kg	SWI: 100133 g/1000 L water POU:100200 g/360 L water	POU, SWI

PREPARATIONS FOR TREATMENT AND PREVENTION OF SKIN DISEASES

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
ECZEKAN 2x8 medication sugar cubes	Dexamethasone- 1 mg, Nicotinamide - 10 mg, Pyridoxine - 50 mg, DL Methionine - 300 mg/8 g	DOG,CAT: 1-5 kg - 1 cube daily; DOG: 5-15 kg: 1 cube daily; 15-30 kg - 1 cube daily; > 30 kg 2 cubes daily. For 4 consecutive days. Subsequent treatment - 8 days, 1/2 dosage.	DOG, CAT

ANTIPARASITIC DRUGS

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
FLUMIXAN 5% 12 kg, 0.6 kg, 10 x 10 g (100 g)	Flubendazole - 5 g/100 g	600 g/1 tonne, with forage	SWI, POU
VERMITAN 20% PREMIX 20 kg, 1 kg, 100 g	Albendazole - 20 g/100 g	SWI: 5 g/100 kg once or 150 g/1 tonne forage for 10 days; SHE, GOA: 2.5-3.75 g/100 kg; CTL: 3.75-5 g/100 kg; HRS: 2.5-3.75 g/100 kg; POU: 600 g/1 tonne forage for 3 days.	SWI, CTL, SHE, GOA, HRS, POU
VERMITAN bolus 600 mg 20 bol.	Albendazole - 600 mg/2.5 g	SHE, GOA: 1/2 bolus/ 40-60 kg; CTL: 1 bolus/60-80 kg	SHE, GOA, CTL
VERMITAN PASTE (HORSES) 15 g	Albendazole - 5 g/15 g	3 divisions/100 kg or 1 syringe / 500 kg	HRS
VERMITAN 10% 100 mL, 1 L, 5 L, 20 L oral suspension	Albendazole - 10 g/100 mL	CTL: 7.5 mL/100 kg; SHE,GOA: 0.5 mL/ 10 kg freely living ruminants (FLR): 710 mL/100 kg	CTL, SHE, GOA, FLR
NEOSTOMOSAN 100 5 mL, 1 L, 5 L perithroid against ectoparasites	Transmix - 50 g Tetrametrin - 5 g/ up to 1 L	CTL, HRS, SHE, PIG, GOA, RBT: 1 L/1000 L water DOG, CAT: 1 mL/200 - 400 mL water	CTL, HRS, SHE, GOA, DOG, CAT, PIG, RBT
POLYVERKAN 1 x 8 medication sugar cubes against all principal endoparasites	Oxybendazole - 40 mg Niclosamide - 200 mg/ up to 8 g	DOG, CAT: 1-5 kg - 1/2 sugar cube; DOG, CAT: 5-10 kg: 1 sugar cube; DOG 10-20 kg: - 2 sugar cubes; DOG 2030 kg: 3 sugar cubes; DOG > 30 kg: 4 sugar cubes. In cases of severe infections, repeated treatmetn after 10 days.	DOG, CAT
DISTO 5 x 25 tablets against fasciolosis and paramphistomatosis	Bithionoloxide - 2 g	1 tablet/50 kg	CTL, SHE, GOA
DOUVISTOME 100 mL, 1 L, 5 L, 20 L oral suspension	Oxyclozanide - 34 mg Methyl parahydroxybenzoate 1.35 mL; Propyl parahydroxy-benzoate - 0.15 mg	CTL: 50-350 kg - 15 mL/50 kg; > 350 kg - 100 mL/ animal; SHE, GOA: < 15 kg - 5mL/animal; 1530 kg - 10 mL/animal;3045 kg - 15 mL/animal; > 45 kg - 20 mL/animal	CTL, SHE, GOA
CESTAL CAT 10 tablets	Praziquantel - 20 mg; Pyrantel pamoate - 230 mg	1 tabl/4 g, incl. pregnant	CAT
CESTAL PLUS 10 tabl, 100 x 2 tabl. against all principal endoparasites	Praziquantel - 50 mg Pyrantel pamoate - 144 mg Fenbendazole - 200 mg	1 tabl/10 kg; young dogs and small breeds: < 2 kg: 1/4 tabl; 25 kg: 1/2 tabl; 510 kg: 1 tabl; medium breeds:1020 kg: 2 tabl; 2030 kg: 3 tabl; large breeds: 3040 kg: 4 tabl; 4050 kg:.5 tabl. and accordingly.	DOG
CEVAMEC 1% 50 mL, 100 mL, 250 mL	Ivermectin - 1 g / 100 mL	CTL, SHE, GOA, SWI: 1 mL/ 50 kg PIG: 1.5 mL/50 kg	CTL, SHE, GOA, SWI, PIG
CEVAMEC 0.6% PREMIX 10 x 10 g, 100g, 0.5 kg, 1 kg, 5 kg, 10 kg, 20 kg	Ivermectin - 6 g/ 1 kg	PIG: < 40 kg: 333 g premix/ 1 tonne forage; 40100 kg: 400 g premix/ 1 tonne forage; SWI: 100 1/2 g ivermectin/ kg mixed with forage for 7 consecutive days or BID at 7-day intervals	SWI, PIG

POLYVITAMINS, AMINO ACIDS AND METABOLISM PROMOTERS

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
SUPRAVITAMINOL LIQ 1 L, 5 L	Vitamins: A, D3, E, B2, B6, B12, PP; Amino acids: methionine, Ca pantothenate, choline citrate, biotin	POU, RBT: 1 mL/5-10 L water; PIG, LMB, GOA: 12 mL/animal; SWI, CLF: 57 mL/animal; SHE: 34 mL/animal; HRS, CTL: 56 mL/50 kg	all species
VIGOSIN 1 L, 5 L	Carnitine, sorbitol, Mg sulfate, enzymes; plant extracts	POU: 12 mL/1 L water; PIG: 12 mL daily CTL, HRS: 2050 mL daily; SHE, GOA: 310 mL/daily; waterfowl: 4 mL/bird.	POU, CTL, HRS, SHE, GOA, PIG
CEVASOL AD3E+C 1 L, 5 L	Vitamins A, D3, E + C	CTL, HRS: 20 mL/animal; CLF, PIG, SHE, GOA: 10 mL/animal the dose is repeated at 2-5 days; POU: 1 mL/5 L water for 5 days	all species

ANTICOCCIDIAL DRUGS

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
VETACOX 25 kg, 1 kg, 100 g	Natrium Sulphadimidin - 80 g Diaveridin - 8 g/100 g	POU, RBT: 1 g /4 L drinking water for 5 days; Ducklings: 1 g /2 L water for 5 days	POU, RBT
CEVAZURIL 100 mL, 1 L, 5 L	Toltrazuril - 2.5 g/100 mL	POU: 28 mL/100 kg/day for 2 consecutive days; 1 mL/1 L drinking water for 2 consecutive days or 3 mL/1 L drinking water for 8 h (single intake)	POU

ANTIANAEMIC DRUGS

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
FERVETRIN 100 mL, 250 mL	Iron Dextran- 200 mg + Phenol - 125 mg/1 mL	CLF: 1 mL/10 kg PIG: 1 mL/animal	PIG, CLF

HORMONAL PREPARATIONS

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
PILLKAN 20 1 x 8 medication sugar cubes for interruption and postponement of the oestrus	Megestrol acetate - 20 mg	CAT, DOG: < 2.5 kg: 1/2 cube; CAT, DOG: 2.5-5 kg: 1/2 cube; DOG, CAT: 5-10 kg: 1/2 cube; DOG: 10-20 kg: 1 cube; DOG: 20-40 kg: 2 cubes; DOG: > 40 kg: 3 cubes (these are daily doses for 3 consecutive days). Following 7 days: 1/2 of the dailty dose	DOG, CAT

POULTRY VACCINES

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
CEVAC Vitapest L 1000 g, 2000 g for vaccination of 1-day old chickens	Newcastle disease vaccine - apathogenic strain	Spray, instillation in the eye - 1-day old chickens; With drinking water - POU > 7-10 days	POU, incl. 1-day old chickens
CEVAC VITABRON L 1000 g, 2000 g	Live vaccine against Newcastle disease and infectious bronchitis. Apathogenic strain +H-120	Spray, instillation in the eye - 1-day old chickens	POU
CEVAC Gumbo L 1000 g, 2500 g	Gumboro vaccine - intermediate type	With drinking water - twice	POU
CEVAC IBD-L 1000 g, 2500 g, 5000 g	Gumboro vaccine - „hot strain"	With drinking water - once	POU
CEVAC TRANSMUN IBD 5000 g, with solvent	Live vaccine against Gumboro „hot strain"	0.2 mL s.c. 1-day old chickens or in ovo	chickens

CEVAC H-120 L 1000 g, 2500 g, 5000 g	Infectious bronchitis vaccine	Spray, instillation in the eye 1-day old chickens; With drinking water: > 7-days	POU
CEVAC BI-L 1000 g, 2500 g	Infectious bronchitis vaccine (B 48) + Newcastle disease (B1)	Instillation in the eye and coarse spray POU < 4-day old; with drinking water - POU > 7-day old	POU
CEVAC MD-HVT 1000 g	Live Marek's vaccine - HVT	0.2 mL i.m./s.c.	POU
CEVAC TREMOR - L 1000 g.	Avian encephalomyelitis live vaccine	Cutaneously, with applicator or with drinking water	POU
CEVAC FP - L with solvent 1000 g	Fowl pox live vaccine	Cutaneously, with applicator	POU
CEVAC UNI L 1000 g, 2500 g	Newcastle disease vaccine HB1	Instillation in the eye and coarse spray - POU < 4-day old; with drinking water - POU > 7-day old	POU
CEVAC NEW L 1000 g, 2500 g	La Sota vaccine	Instillation in the eye and with drinking water	POU
CEVAC NEW K 1000 g	Inactivated Newcastle disease vaccine	0.5 mL s.c. at the age of 16-20 weeks	HEN
CEVAC ND-IB-EDS-K 1000 g. Combined vaccine	Inactivated combined vaccine against Newcastle disease, infectious bronchitis and egg-drop syndrome Inactivated Newcastle disease vaccine	0.5 mL s.c. at the age of 16-20 weeks	HEN
CEVAC SET K 1000 g	Inactivated combined vaccine against Salmonella	0.5 mL s.c. at the age of 14-16 weeks	HEN
CEVAC ND IB IBD EDS K 250 mL, 500 mL	Polyvalent inactivated vaccine against Newcastle disease, IB, Gumboro and egg-drop syndrome	0.5 mL/ bird s.c. or i.m. at the age of 16-20 weeks or 3-4 weeks prior to beginning of egg laying	POU-breeders, broilers, layer hens
CEVAMUN 1 tablet for dechlorination, pH regulation and water marking for live vaccines	S. thiosulfate, Patent Blue V	1 tablet/100 L water	For all live poultry vaccines

COCCIDIOSTATIC FOR BROILERS

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
AVIAX 5% x 25k g	Semduramycin 50g/1kg	0.5 kg/1 tonne forage (25 ppm)	Broilers

ANTIMYCOTIC DRUGS

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
MYCOTOX NG 1 kg, 5 kg, 25 kg premix forage supplement	Thymol - 50 g/kg micronized brewer's yeast	0.5 kg/tonne for slightly to moderately contaminated forage; 1-3 kg/tonne for severely contaminated forage	POU, SWI

VACCINES FOR SHEEP AND PIGS

PRODUCT	ACTIVE SUBSTANCE	DOSAGE	ANIMAL SPECIES
COGLAMUNE 50 mL 100 mL, 250 mL	Inactivated vaccine against necrotic enteritis Cl. perfringens type A, C and D	PIG, SHE, GOA, CLF: < 200 kg - 2 mL/ animal; SWI: 4 mL/ animal	SWI, PIG, SHE, GOA, CLF
COGLAVAX 50 mL, 100 mL, 250 mL, 500 mL	Polyvalent inactivated vaccine agaisnt enterotoxaemia sheep, anaerobic dysentery, haemorrhagic enteritis lambs; soft nephropathy, Bradzot, necrotic hepatitis, tetanus, postparturient gangrene	2 mL /sheep	SHE, LMB

QD - every day; BID - twice a day; CTL - cattle; HRS - horse, SHE- sheep, GOA - goat, RBT - rabbit, LMB - lamb, CLF - calf, FIS - fish, SWI- swine, POU - poultry, FLR - freely living ruminants;
☐ preparations applying for registration are given;